



















# BRAIN :

A JOURNAL OF NEUROLOGY.

VOL. XIII.





*Ms. B.*

# BRAIN :

A JOURNAL OF NEUROLOGY

EDITED

FOR THE NEUROLOGICAL SOCIETY OF LONDON

BY

A. DE WATTEVILLE

VOL. XIII.

*30195  
8/11/93.*

London:  
MACMILLAN AND CO.  
AND NEW YORK.  
1890.

RC  
321  
B7  
v. 13

LONDON:  
PRINTED BY JOHN BALE AND SONS,  
87-89, GREAT TITCHFIELD STREET, W.

# CONTENTS.

—o—

## ORIGINAL ARTICLES:—

	PAGE
On the Simulation of Hysteria by Organic Disease of the Nervous System. By Thomas Buzzard, M.D., F.R.C.P. ... ..	1
Of the Visual Area of the Cerebral Cortex, and its Relation to Eye Movements. By Professor Hermann Munk (Berlin) ... ..	45
A Contribution to the Pathological Anatomy of Chorea with the Report of a Case. By Charles L. Dana, M.D. (New York) ...	71
Notes of a Case of Tumour of the Cerebellum with an Absence of all Symptoms. By P. W. Macdonald, M.D. ... ..	83
Case of Hæmorrhages in and about the Pons. By J. S. Risien Russell, M.B., and James Taylor, M.A., M.B. ... ..	88
The Psycho-Physical Process in Attention. By J. Sully, M.A. ...	145
On Associated Eye-Movements produced by Cortical Faradization of the Monkey's Brain. By F. W. Mott, M.D., and E. A. Schaefer, F.R.S. ... ..	165
On Movements resulting from Faradic Excitation of the Corpus Callosum in Monkeys. By F. W. Mott, M.D., and E. A. Schaefer, F.R.S. ... ..	174
A Contribution to the Study of Cheyne-Stokes' Breathing. By J. Dixon Mann, M.D., F.R.C.P. ... ..	178
Treatment by Suspension. By J. S. R. Russell, M.B., and James Taylor, M.A., M.B. ... ..	206
Preliminary Note on certain Morbid Products found in the Brains of Patients Dying after severe Head Injuries. By Alexander Miles, M.B., L.R.C.S. (Edinburgh) ... ..	224
Syringomyelia. By Dr. Paul Blocq ... ..	289

	PAGE
On Disorders of the Musical Capacity from Cerebral Disease. By A. Knoblauch, M.D. ... ..	317
On the Pathological Value of the Gasserian, Lenticular, Spinal and Cardiac Ganglia. By W. Hale White, M.D. ... ..	341
Remarks on Mr. Sully's Paper on the Psycho-Physical Process in Attention. By Prof. A. Bain (Aberdeen) ... ..	348
The Bi-Polar Cells of the Spinal Cord and their Connections. By F. W. Mott, M.D.... ..	433
The Effect of Movements of the Human Body on the Size of the Spinal Canal. By R. W. Reid, M.D., F.R.C.S., and Ch. S. Sherrington, M.A., M.B. ... ..	449

## CLINICAL CASES:—

A Case of Bulbar Paralysis without Structural Changes in the Medulla. By Lauriston E. Shaw, M.D., M.R.C.P. ... ..	96
A Case of Poliomyelitis Chronica occurring in a Child aged Five Years. By F. W. Kirkham... ..	100
A Case of Hemiatrophy of the Tongue with its Pathology. By E. F. Trevelyan, M.D. (Lond.), B.Sc. ... ..	102
Case of General Paralysis Complicated by Aphasia. By C. Price Tanner ... ..	111
Complete Paralysis of Right Third Nerve in a Patient affected with Lead Palsy—Recovery under the Direct Application of Galvanic Currents. By Thomas Buzzard, M.D., F.R.C.P. ... ..	227
Paralysis especially of One Deltoid Muscle in a Patient Suffering from Lead Poisoning; Preserved Electrical Reaction of the Muscles. By Thomas Buzzard, M.D., F.R.C.P. ... ..	234
A Case of Diphtheria with Absence of Knee Jerks. By Seymour J. Sharkey, M.D., F.R.C.P. ... ..	237
On a Case of Ataxic Paraplegia with Autopsy, and a Case of Locomotor Ataxy; Suspension; Death from Septicæmia; Autopsy. By J. Michell Clarke, M.A., M.B. (Camb.), M.R.C.P. (Lond.)... ..	356
Acute Ascending Paralysis (Landry's Disease), followed by Ataxic Paraplegia. By Sanger Brown, M.D. ... ..	375

On Paralysis of the Third Nerve as a Complication of Graves' Disease. By James Finlayson, M.D. ... ..	383
Notes of a Case of Progressive Nuclear Ophthalmoplegia. By W. M. Beaumont ... ..	386
A Case of Spastic Spinal Paralysis—Treatment—Recovery. By E. Dutoit, M.D. (Berne) ... ..	389
Notes on Three Cases of an Hereditary Form of Progressive Amyotrophy. By H. B. Donkin, M.B. (Oxon.), F.R.C.P. ... ..	456
A Case of Recovery from Cerebro-Spinal Meningitis. By W. Hale White, M.D. ... ..	461
Paralysis Agitans in a Young Man. By W. B. Hadden, M.D., F.R.C.P. ... ..	465

## CRITICAL DIGESTS:—

Chronic Hydrocephalus. By M. Armand Ruffer, M.A., M.D. ... ..	117, 240
Friedreich's Disease. By Dr. P. Ladame ... ..	467

## REVIEWS AND NOTICES OF BOOKS:—

Lewis : A Text-book of Mental Diseases ; with Special Reference to the Pathological Aspects of Insanity. By John Batty Tuke ... ..	270
Bowlby : Injuries and Diseases of Nerves and their Surgical Treatment. By Herbert W. Page ... ..	279
Steiner : The Functions of the Central Nervous System and their Phylogenesis. By F. W. Mott ... ..	391
Falret : Etudes Cliniques sur les Maladies Mentales et Nerveuses. By E. Birt ... ..	398
Adenot : Des Méningites Microbiennes. By M. Armand Ruffer ... ..	405
Sibut : De l'Atrophie Cérébrale Partielle d'Origine Périphérique. By M. Armand Ruffer... ..	406
Clevenger : Spinal Concussion. By William Thorburn ... ..	408

	PAGE
Obersteiner : The Anatomy of the Central Nervous Organs in Health and in Disease. By A. Beevor, M.D. ... ..	538
Edinger : Twelve Lectures on the Structure of the Central Nervous System. By A. Beevor, M.D. ... ..	541
Mercier : Sanity and Insanity. By James Anderson, M.D. ...	542
Souza-Leite : De l'Acromégalie, Maladie de P. Marie. By M. Armand Ruffer, M.D. ... ..	544
Luys : Small Photographic Atlas of the Nervous System. By P. Chaslin, M.D. ... ..	547

#### ABSTRACTS OF BRITISH AND FOREIGN JOURNALS :—

Miscellaneous Abstracts ... ..	284, 422
Current Nerve Anatomy and Physiology. By Alex. Hill, M.D. ...	550

## LIST OF CONTRIBUTORS.

—o—

- |                                 |                                  |
|---------------------------------|----------------------------------|
| ANDERSON, JAMES, M.D.           | MANN, J. DIXON, M.D., F.R.C.P.   |
| BAIN, PROF. A. (ABERDEEN).      | MILES, ALEXANDER, M.B., L.R.C.S. |
| BEAUMONT, W. M.                 | (EDIN.)                          |
| BEEVOR, A., M.D.                | MOTT, F. W., M.D.                |
| BIRT, E., L.R.C.P., M.R.C.S.    | MUNK, PROF. HERMANN.             |
| BLOCQ, DR. PAUL.                | PAGE, HERBERT W., M.A., F.R.C.S. |
| BROWN, SANGER, M.D.             | REID, R. W., M.D.                |
| BUZZARD, THOMAS, M.D., F.R.C.P. | RUFFER, M. ARMAND, M.A., M.D.    |
| CHASLIN, P., M.D.               | RUSSELL, J. S. RISIEN, M.D.      |
| CLARKE, J. MICHELL, M.A., M.B.  | SCHAEFER, A., F.R.S.             |
| (CAMB.), M.R.C.P. (LOND.)       | SHARKEY, SEYMOUR J., M.D.,       |
| DANA, CHARLES L., M.D. (NEW     | F.R.C.P.                         |
| YORK).                          | SHAW, LAURISTON E., M.D.,        |
| DONKIN, H. B., M.B. (OXON.),    | M.R.C.P.                         |
| F.R.C.P.                        | SHERRINGTON, S., M.A., M.D.      |
| DUTOIT, E., M.D. (BERNE).       | SULLY, J., M.A.                  |
| FINLAYSON, JAMES, M.D.          | TANNER, C. PRICE.                |
| HADDEN, W. B., M.D., F.R.C.P.   | TAYLOR, JAMES, M.A., M.B.        |
| HILL, ALEXANDER, M.D.           | THORBURN, WILLIAM, F.R.C.S.      |
| IDELSON, VALERIUS, M.D.         | TREVELYAN, E. F., M.D. (LOND.),  |
| KIRKHAM, F. W.                  | B.Sc.                            |
| KNOBLAUCH, A., M.D.             | TUKE, JOHN BATTY, M.D., F.R.C.P. |
| LADAME, P., M.D. (GENEVA).      | (EDIN.)                          |
| MACDONALD, P. W., M.D.          | WHITE, W. HALE, M.D.             |





## LIST OF ILLUSTRATIONS.



	PAGE
The Visual Area of the Cerebral Cortex, and its Relation to Eye Movements, Figs. 1-4      ...      ...      ...      ...	64
The Pathological Anatomy of Chorea ...      ...      ...	80
Hæmorrhages in and about the Pons, Figs. 1-3      ...      ...	95
Hemiatrophy of the Tongue ...      ...      ...      ...	104
Associated Eye Movements produced by Cortical Faradization of the Monkey's Brain      ...      ...      ...      ...	168
Diphtheria with Absence of Knee Jerks      ...      ...      ...	237
Disorders of the Muscular Capacity from Cerebral Disease, Figs. 1-8      ...      ...      ...      321, 326, 327, 328, 329, 334, 338, 339	
Ataxic Paraplegia. Locomotor Ataxy, Figs. A. to H. 371, 372, 373, 374	
Spinal Bipolar Cells (Photogravures), Figs. 1-6      ...      ...	448
"          "      (Lithographic Plates), Figs. 1-5      ...      ...	448
The Effect of Movements of the Human Body on the Size of the Spinal Canal      ...      ...      ...      ...      ...	452



# BRAIN.

PART I., 1890.

## Original Articles.

### ON THE SIMULATION OF HYSTERIA BY ORGANIC DISEASE OF THE NERVOUS SYSTEM.<sup>1</sup>

BY THOMAS BUZZARD, M.D., F.R.C.P.

IN our practice as physicians we are continually meeting with cases which suggest the presence of organic lesion of the nervous system, whilst, at the same time, circumstances occur to make us doubt whether the symptoms present may not be dependent upon what, for want of a better term, is styled functional disorder. It is quite unnecessary here to attempt to define what is meant by the term, nor to insist upon the importance, both as regards prognosis and the treatment of the condition, that our diagnosis should be correct.

The field occupied by cases of this description is so vast that it would be ridiculous for me to attempt anything like a survey of it. I propose therefore on this occasion to limit my observations to those instances in which some loss of power in a limb or limbs is the dominating feature. The admirable paper which was recently read before this Society by Dr. Hughes Bennett on "Muscular Hypertonicity" will be very present to your minds as discussing those numerous examples which wear the semblance of spastic paraplegia, and with these therefore it will be unnecessary for me to deal. Nor

<sup>1</sup> Presidential Address at the Annual Meeting of the Neurological Society of London on January 23rd, 1890.

shall I dwell much upon the more common forms of so-called hysterical paralysis, the recognition of which the surrounding circumstances may render very easy. In fact I cannot hope to do more than discuss a few points of interest and difficulty, without pretending to any attempt at exhausting an exceedingly wide subject.

One difficulty in connection with the class of cases to which I refer lies in the fact that under the term functional we probably have often to include (because of the impossibility of differentiating them) examples of various forms. There is, in the first place, involuntary simulation of organic disease associated with some morbid psychical condition to which the term hysterical is usually applied. Next, there are examples of functional inability dependent probably upon a temporary malnutrition of certain parts of the nervous centres. There appears reason to believe that this local imperfection may sometimes exist for a long while without giving rise to destructive changes in the parenchyma, but that in many instances it represents but a stage of actual change in tissue which eventually becomes permanent and irrecoverable. How far we may reckon that there is a point in this progressive deterioration at which treatment may be successfully applied must still be considered doubtful, although evidence tends rather to the idea that this is probably the case. The cases of this class present peculiar difficulty in their diagnosis, because, combined with evidence of local troubles of nutrition in the nerve centres, there are often so many symptoms of hysterical or emotional character as to greatly obscure and confuse the picture.

It is especially to this category that I propose to draw attention on the present occasion. I fear that my remarks will necessarily tend rather to expose than to solve the difficulties which we are liable to encounter in dealing with this question, and that their discursive character may suggest, what is indeed the truth, that I am rather employed in thinking aloud than in imparting information. As far as possible I shall relate the sequel of cases to which reference is made. This will often decide a question which has been a puzzling one. Owing to the kindness of my friend, Dr.

Playfair, with whom I have examined during the last ten years many cases in which a serious question of diagnosis has arisen, I shall be able to refer to the results of the Weir Mitchell treatment applied with his well-known skill. The physician truly experiments, as Claude Bernard has said, when he tries to bring about a modification in the symptoms of disease. In some cases, to which reference will be made, the disappearance under improved nutrition of the symptoms which gave rise to the doubt has resolved the difficulty in the happiest way possible. In others the question has been determined by failure to interrupt the course of the malady, or by a fatal issue.

There is a form of paraplegia of which I have seen several examples, in nearly all of which important questions have arisen as to the functional or organic nature of the condition. The subject will be best introduced by briefly relating particulars of a few cases.

A young lady, æt. twenty-one, was seen by me in consultation with Dr. Playfair on November 1st, 1886.

Since the age of fifteen she had been observed by her friends to have a peculiar gait, and this was noticed two years later by a surgeon who was consulted on account of some pain in her joints. At that time she walked with a stiff and stilted gait, which was chiefly noticeable in going upstairs, and varied very much. There appeared, however, I was told, to be no want of muscular power. She could walk well, and dance. From time to time she suffered from a variety of typical hysterical symptoms, including an aversion from food, under which she had greatly lost flesh and strength at the time that I saw her. It was especially during several preceding months that she had gradually failed in her walking powers, and on several occasions had fallen down.

The question to be determined was whether the condition was an hysterical one, as it had been pronounced to be by more than one medical authority, and likely therefore to yield to a course of Weir Mitchell treatment, for which purpose she had been taken to Dr. Playfair.

On examination I observed that the girl was highly anæmic. She could stand or walk without support, but

slowly and laboriously, inclining the trunk to the side opposite to that from which depended the limb which was being advanced. When in the recumbent position she could flex the thighs upon the trunk with a fair amount of force, but when seated she had little or no power of lifting either knee. Extension at the knee joint was only moderately well performed, whilst the power of flexion at the same joint, and extension and flexion at the ankle joint, appeared to be normal. When standing it was with great difficulty that she could place her right foot upon the sofa, and only by adopting a swinging movement. She could not even swing the left foot on to it. With her right foot on the sofa she could not lift herself up so as to stand upon it. The muscles of the lower extremities were well developed. There were no sensory symptoms. No complaint was made of the upper extremities.

The wrist jerks were exaggerated; the knee jerks, on the other hand, were decidedly feeble. This contrast in the state of the deep reflexes, when taken in conjunction with the other symptoms, gave important evidence. I have often referred to the fact that the knee jerk is not, according to my observation, lost in cases of hysteria; on the contrary, it is almost always increased. Its marked diminution in this case, alongside of greatly exaggerated wrist jerk, pointed in the circumstances to impaired nutrition of the second lumbar segment of the spinal cord. The complete loss of power in the iliaco-psoas muscles, as shown by the examination of the patient's movements, indicated that the first lumbar segment was still more severely affected. An opinion to this effect was given. As regards the Weir Mitchell treatment, it was agreed that the general health was likely to receive benefit from it, but that no promise could be made that the local symptoms would be removed.

The patient went through a course of systematic treatment conducted by Dr. Playfair, who at the end of six weeks was kind enough to let me again examine her. She was no longer anæmic, and had greatly improved in appearance. She had gained fifteen pounds in weight. The knee jerks were now well marked, but there was still considerable

difficulty in the movements performed by the iliaco-psoas muscles. She could "fling" her foot on to a low chair—it could scarcely be called lifting it—and could then drag herself up, holding on to the back of the chair, so as to stand upon the seat. This was only when the right foot was employed.

I am informed that this lady still shows a somewhat peculiar gait, but that she can dance, run about quite actively, ascend stairs quickly, and appears to have full muscular power. She was recently married.

Some four years previous to this I had been consulted in the case of a well-nourished healthy-looking young woman, æt. twenty-four, who had failed in her walking powers, and a similar question had arisen as regards the nature of her disability. It is notorious that when a girl of highly neurotic temperament complains of difficulty in walking, the suggestion of an hysterical cause is very apt, and in many cases justly, to arise.

The patient, whom I saw in 1882, had been observed by her mother in 1879 to be walking in a laboured way, which was at first attributed to the wearing of high-heeled boots, and the presence of chilblains. But the difficulty continued and only increased as time went on. It appeared that in the winter of 1876-77 she had undergone great fatigue in "rinking," and it was not long after this that she had begun to feel unduly tired in walking.

I found on examination that the muscles of the lower extremities were well developed, and that the only defect in movement was in the action of the iliaco-psoas group. There appeared to be absolutely no power of flexing the thigh upon the pelvis, all the other movements being well performed. The knee jerks were normal. The patient was sent to bed, fed, rubbed, and fired in the lower dorsal region of the spine, but without any effect. In December 1883 she could still walk, but required first to be set upon her feet.

I learned last year that she had lost all power of movement in her lower extremities. There was no wasting of the limbs, which were sound and indeed unduly firm.

Last year a sister of this patient, æt. twenty-eight, was

brought to me suffering in her turn from a somewhat narrowly localised loss of power. This patient could run up stairs two steps at a time, and mount easily upon a chair without the aid of her arms, but she had no power of dorsal flexion of the right foot, and almost none in the left. The leg itself was very firm and plump. Electrical examination shewed complete absence of reaction to induced currents in the anterior tibial muscles.

The patient, I learned, had enjoyed very good health, but her walk had been observed to be peculiar for eight or nine months. This was due, no doubt, to the dropping of her feet necessitating high action. Besides the patient with iliaco-psoas paralysis above described, this girl has two other sisters, one whom I have not seen, who is thirty-two years old and married, and is described as walking in the same fashion. She is unable to walk fast. The other sister, twenty-five years of age, shows no peculiarity of gait. It is evident that in the first of these two sisters' cases no question as between the organic or functional nature of the affection could have arisen, had the muscles affected (the flexors of the thigh) been in a position to be explored by electric currents as they were in the second. But although in very emaciated persons it may, though very rarely, be possible to apply a rheophore in the situation of the iliacus muscle, the pain caused by electric currents in this situation is intolerable to most persons, and renders electrical exploration impracticable.

I suppose it can hardly be doubted that the condition of the anterior tibial muscles, in the second sister, gives the clue to that of the iliaco-psoas muscles in the first, and from the description given of the married sister (whom I have not seen) it seems likely that she is also affected in some of her muscles in a similar manner. It seems probable that these are cases of simple idiopathic muscular atrophy which is apt to occur in several members of the same family, is often inherited, and is not of central origin. The remarkably firm condition of the limbs, coupled with the results of electrical examination, seems to point to overgrowth of interstitial tissue. In this family I could hear of no other instance of a like affection, and the only peculiarity in the history that



I could learn was that the father and mother were first cousins.

Some years ago I saw with Dr. Playfair another lady who had been affected for more than ten years with a similar powerlessness confined to the iliaco-psoas muscles. In her case the Weir Mitchell treatment was adopted without success. She was probably an example of the same condition.

In 1885, a military man, æt. sixty-eight, consulted me on account of inability to flex the hip joints—the other movements of the lower extremities being perfect, and the electrical reactions normal. There was no loss of sensation. The knee jerks were perfect, and there was no difficulty in the action of the bladder. The want of power first showed itself at the close of the Crimean campaign, during which he had enjoyed good health. He was affected with peculiar nervous sensations. In this case there was a history of syphilis. Specific treatment had produced no effect.

Atrophy of the iliaco-psoas muscles occurring in a young female, accompanied by hysterical symptoms, would seem to be peculiarly liable to be wrongly interpreted. The patient complains of difficulty in walking, but the knee jerks are preserved, and the electrical condition of all the muscles of the lower extremity that can be tested is strictly normal. One is naturally disposed to expect in such a case that if there be a central lesion in the cord, either the muscles innervated from the lumbar enlargement will be found atrophied and their electrical reactions abnormal, or if the lesion be above the enlargement, symptoms of spasticity will be present, the knee jerks will be exaggerated, and there will be more or less ankle clonus.

I think that the cases which I have described may serve a useful purpose in drawing attention, in a doubtful case, to the fact that there may be isolated atrophy of the iliaco-psoas.

The successful result of systematic treatment by high feeding and massage in the case first related is probably to be explained by the fact that the patient was anæmic and much reduced. It seems likely that in her case the condi-

tion was not one of congenital idiopathic muscular atrophy, but that there was a temporary state of mal-nutrition of the cord, which recovered. I remember another instance of the kind in a young woman profoundly affected with anæmia, who regained power under appropriate treatment.

A governess, æt. twenty-three, came to me on Oct. 18th, 1884, on account of difficulty in walking. She had been delicate in childhood, but had usually enjoyed good health. For the last fortnight she had been unable to carry on her occupation. Five weeks previously she had felt her breath very short in going up stairs, and had kept in bed for a few days. Every time she left the bed she fainted. A fortnight since she experienced great difficulty in walking, as though her legs gave her no support, and there was a great aching at the bottom of the back. She could only just hobble about from one room to the other. There was great vesical irritability. The catamenia had been suspended for three months. The patient was suffering from marked chloro-anæmia; the cardiac sounds were normal; the urine contained no albumen nor sugar. There was some tenderness on pressure on the first lumbar vertebra; the spinal column showed no deformity. The movements of the lower extremities were feeble, especially flexion of the thigh upon the hip, which could scarcely be performed. The knee jerks were rather in excess. Iron and arsenic were prescribed for her.

After a fortnight she had greatly improved in colour and was able to walk better. The vesical trouble, however, still continued. She was unable to lift the left knee, but could lift the right. There was no affection of sensibility. In three weeks more she felt quite well in herself and the vesical trouble had ceased. She was able to lift the right knee against a certain amount of pressure, but not the left at all. She could straighten the left leg, flex and extend the foot, and in fact do anything but lift the knee. In going up stairs she would put her right foot down first, and then draw the left after her; in descending the left was put down first. She still suffered from palpitation and breathlessness.

About six weeks later, when I saw her again, the legs

had quite recovered; she was able to walk more than a couple of miles, and could lift her knee on either side in normal fashion.

It will be remembered that the late Dr. Moxon, in his Croonian Lectures before the Royal College of Physicians in 1881, called attention to the great length and obliquity of the spinal arteries which supply the lower third of the spinal cord, as a probable cause of temporary anæmia. It would seem that where there is general anæmia this anatomical arrangement may explain the localisation of disturbed nutrition in this part. I suppose that in the case first related, where the knee jerks, which were at first small, became well pronounced as the patient recovered, it was to the anæmic condition of the cord at the second lumbar segment that the imperfection of the reflex action was due. One can conceive it possible that anæmia in a given case might be so extreme as to cause a temporary disappearance of the reflex. It is necessary to bear this in mind, as the absence of knee jerk is a symptom which, if the illness has been long continued, is more suggestive of irreparable damage than of a condition capable of being treated successfully.

The absence of knee jerk, so far as my experience goes, in a young female is a matter requiring very careful examination. I need not mention the obvious conditions which usually explain the absence, but would refer to cases of a less common kind, which it is well to bear in mind. One of these I saw with Dr. Playfair in 1884. The patient, a young lady, aged twenty-six, had begun to get weak in walking eight or nine years previously. The difficulty had gradually increased until she had an attack of typhoid fever, since when she had been unable to walk at all.

Examination showed the following condition:—Her speech is ataxic, the words being slurred. The hands possess a good grasp, but are rather wildly moved about when directed to touch an object—a condition of marked ataxy. This is the case with each hand. It is not the tremor of disseminated sclerosis, but the movement of a person suffering from *tabes dorsalis*. There is no wrist jerk on either side. She cannot sit up. When Dr. Playfair and I tried to

make her stand and walk between us, she could do nothing ; her legs and feet disappeared and were mere appendages to her frame. There was no power whatever of standing, she sank "in a heap" to the ground. Lying on the couch, she failed to lift the right foot from off the ground and place it on the couch. The sensibility of the limbs appeared good. The knee jerks were absolutely wanting; the vasti interni muscles were flaccid and did not respond to direct percussion; the sole reflex was preserved. There was enormous lateral curvature of the spine. This, she informed me, had not been observed until she was ten years of age. There had never been any pains. The powerlessness of the muscles of the trunk and lower extremities was very great. With all this the action of the bladder and bowels was described as normal, although there had been occasional incontinence.

The patient had a fairly good colour with a face not markedly unhealthy looking. Her manner was highly nervous and hysterical. The pupils responded well to light.

It appeared that her condition had been ascribed to hysteria, and that in an earlier stage of her illness she had been forced to try and walk up stairs. She managed this only by the aid of the banisters, never without. At that time (three years previously) she could get about a room with the help of a chair. The general health was good. I was of opinion that the case was one of Friedreich's ataxy and that it was not fitted for the Weir Mitchell treatment.

Her mother tells me that she has two sons and one daughter besides the patient. They are in very good health, able to play lawn tennis and take long walks. She herself enjoys good health, and so does the father of the patient. Neither in her family nor in her husband's is there any case of paralysis or nervous disorder so far as she knows.

As is well known, Friedreich's ataxy is usually not confined to one member of a family, so that in this instance the exceptional limitation increased the likelihood of the case being referred, as indeed it had been positively, to hysteria; but there can be no question of its nature.

A year or two ago a young man, æt. twenty-four, was brought to me on account of difficulty in walking, the cause

of which had given rise to difference of opinion. He had a staggering gait, with hesitating and slurring speech. The knee jerks were absent.

It appeared that up to fourteen months old he had been liable to "spasmodic croup," and he was two and a-half years old before he began to stand upon his feet. As a school boy he would tumble about at times and never could learn to dance. He did well, however, in his school work, and carried off prizes, the mental development being good. He went to one of the universities, but had to quit it after two years, the proctors accusing him of intoxication, owing to his gait. This was, evidently, a case of Friedreich's ataxy, but here again I could obtain no history of a family proclivity to the disease. There was an elder brother and a younger sister, both of whom I was informed were free from disease, and I could not hear of any other member of the family having been affected in a like manner.

I cannot remember (and my very imperfect notes say nothing upon the point) whether either of these patients presented the deformity of foot which has been specially described by Rutimeyer, Burg and Ormerod. My attention was first drawn to the symptom by Dr. Ormerod, who thus describes the development of the kind of club foot which is apt to occur in cases of Friedreich's ataxy. "The instep becomes prominent, and the meta-tarsals appear to be shortened; at the metatarso-phalangeal joints the toes become over-extended, and at the first inter-phalangeal joints flexed. The foot looks humpy and shortened. Below, the plantar arch (at least as seen from the inner side), is abnormally high, and the balls of the toes are very prominent. The foot also tends to assume the position of equino-varus."<sup>1</sup>

I remember seeing a case (which I do not now doubt was one of Friedreich's ataxy, although I failed to diagnose it at the time), in which the only complaint made by the patient was of this club foot, to which was attributed the difficulty in gait. The knee jerks were absent. It may be well to mention this, as there is always the possibility of the disease

<sup>1</sup> BRAIN, January, 1888.

being overlooked from the attention being directed to one prominent symptom.

There is no doubt that as a rule the fact that a patient has been observed on some occasion when she thought herself alone, to perform some movement which has been previously alleged to be impossible, lends us very important aid in coming to a diagnosis. This circumstance occurred in a case which I will briefly relate.

A lady, aged thirty-three, was seen by me at Dr. Playfair's request on June 16th, 1884. She had been for nearly eighteen years confined to her bed on account of loss of power in her lower extremities.

The patient had fallen down a flight of steps at the age of fourteen or fifteen, and shortly afterwards began to walk with difficulty and only with the help of sticks and crutches. She was seen by an eminent surgeon, who thought that she might have injured her spine and advised that she should rest. She took to her bed, and shortly afterwards completely lost the use of her lower extremities.

The patient when I visited her lay on a couch, with her legs semi-flexed at the knees and turned on their side. The feet were dropped and toes pointed. She said that she was unable to move her legs voluntarily in the least, but that they moved sometimes of themselves. She knew how they were lying without looking at them. The muscles when examined electrically were found to contract normally to induced currents. There was complete anæsthesia of both legs from just above the knee downwards—in fact of that portion of the lower extremity which would be covered by a stocking. The limbs were emaciated, the muscles being small and flabby, but there was no picked-out atrophy. The knee jerk was good on each side, and the tendo achillis jerk well marked; there was no ankle clonus. The plantar reflex could scarcely be evoked. The spinal column was free from deformity, there was superficial tenderness in the mid-dorsal region, and also in the nape of the neck. The patient gave me her left hand saying that the right was somewhat weak. The muscles of that arm contracted normally to induced currents.

The cutaneous anæsthesia was entirely confined to the legs. The patient did not suffer from any pains in the extremities ; there were no bed sores. She was liable to severe dysmenorrhœa. The sight was perfect, the intelligence high, the general health very good.

I was informed that on two or three occasions the urine had been passed in the bed without the patient's knowledge. On one occasion when she had been laid up for about ten years, her maid, coming unexpectedly into her room, found her standing at the window, whence she walked to bed again. Even without this bit of history the case, it will be seen, presented no difficulty in diagnosis.

A confident opinion was given that the case was one of hysteria, and the patient was put through a course of systematic treatment. She perfectly recovered. Dr. Playfair informs me that she has remained well ever since, and leads a very busy and energetic life.

A recent experience reminds me that we must be cautious in drawing inferences from some behaviour of the patient which appears inconsistent with other symptoms. On the 8th November last I saw in consultation a lady, æt. fifty-five, of highly nervous temperament with hysterical antecedents who had been suffering for several days past from attacks of violent colic-like abdominal pain, extending at times to the lower extremities. She described the attacks as commencing with a "creeping" up the spine. In the course of time there would be a state of intense excitement with convulsive movements of the arms.

It appeared that one of her breasts had been removed in the early part of the year on account of malignant disease, and the patient was under so fixed a belief that the present illness signified a reappearance of this disease, that she could not be pacified.

The patient looked thin, but not emaciated. She lay in bed and appeared to have especially great difficulty in flexing the thighs upon the trunk ; dorsal flexion of the feet was performed more easily. The muscles were very flabby. The knee jerks were absent, and there was no plantar reflex. She complained of numbness over both lower extremities



from about the level of the tenth dorsal vertebra downwards, and in this district touches were felt imperfectly. She told me that a fortnight previously numbness had commenced in the right lower extremity, and also rather to the left of the spine. At the same time there was weakness of the right leg. This had gradually, within the last few days, invaded the left lower limb, but the right was still the worst. She was sure that the loss of power and the numbness both commenced together in the right limb. Nothing wrong was to be detected about the spine nor in the abdomen.

The nurse said that the patient when by herself walked about the room quite actively, in strong contrast to the feebleness of movement shown when she was observed. In view of this, the patient's manner and history, and the absence of any sign of growth under the most careful examination, opinions had been expressed that the symptoms were probably functional, and it was with a view of helping to clear up this initial difficulty that my opinion was asked. From the fact that, whilst the wrist jerks were strongly marked (as is common in hysterical women), the knee jerks were quite absent, I had no difficulty in coming to a conclusion that the symptoms depended upon organic lesion. The opinion was expressed that she was suffering from neuritis, and this was assigned to the cauda equina. It might be due either to rheumatism, gout, syphilis, or malignant disease. The latter contingency was to be feared in the circumstances. Large doses of iodide were advised.

On Nov. 20th, there was still further loss of power, but the attacks of colic-like pain had ceased. The vasti muscles did not respond to induced currents. On Dec. 6th there was absolute paraplegia except that she could just move the toes of the right foot. The bladder was not affected. There was difficulty in swallowing and dangerous choking. Her condition became more and more grave, and on Dec. 9th she died, after an attack of choking.

There can be no doubt that she succumbed to secondary deposits of cancer.

In the case preceding that which has just been related, it will be noted that the patient had occasionally suffered



from unconscious discharge of urine. In my experience this is not at all of unfrequent occurrence in cases of hysterical paraplegia. The behaviour of the bladder has sometimes been made a crucial test in the question between functional and organic disease. It is quite certain that patients affected with hysterical paraplegia may suffer from inability to prevent discharge of urine into the bed. It is notorious, of course, that they are liable to retention. An instructive illustration of urinary troubles occurring in the course of a case which was evidently hysterical will be found in the narrative which follows.

A female patient, æt. thirty-three, was admitted into the National Hospital for the Paralysed and the Epileptic on May 26th, 1886, on account of loss of power in both legs and the left arm, of five years' duration.

In the autumn of 1879 the patient had suffered from "inflammation of the lungs," and afterwards from general weakness with "weak heart" and faintness. In 1881 her legs became very weak, and at the end of the year she could not move them. In 1883 the legs began to get stiff and since then they had gradually become worse.

In 1864 she had fallen downstairs and hurt the left side of the head. This was followed by a discharge from the left ear, and about a year later an abscess formed behind it, and there had been more or less discharge from the abscess or the ear ever since.

In 1878 she had broken "her left arm and wrist and finger," so she described it, and the hand had been useless ever since. The arm, however, and the elbow and shoulder had only been helpless since last July.

She never had severe pain in the back or limbs, and had never suffered from bed sore. There had been incontinence of urine for some months during the past year; constipation had existed since 1869. There had not been any loss of control over the sphincter ani.

The patient had suffered from scarlet fever at twelve, and diphtheria at seventeen. As regards her family history, the father as well as two of his sisters and two of his brothers and many other relations had died of consumption. Her

mother had cancer ; many of her relations have had paralysis.

On admission the following note was taken by the house physician. There is complete left facial paralysis ; a foul discharge comes from the left ear, copious in amount, with tenderness on pressure over the mastoid process, where there is a mark of an old scar. A short distance inside the external auditory meatus a pale polypoid mass is seen. In the left eye corneal opacities are seen in the lower half. There is no paralysis of the tongue, no difficulty in articulation or deglutition. There is aphonia : attempts to examine the larynx failed.

The left hand lies in a position of rigid flexion, the joints cannot be voluntarily moved. The patient cannot stand ; there is rigidity of the legs in the extended position. Both ankles are extended and the toes over-extended on the metatarsus. There is no voluntary power over the joints. Knee jerks are equal ; no ankle clonus can be produced, possibly on account of mechanical impediment ; the wrist jerks are equal. There is no affection of sensation anywhere. The plantar reflexes are present, though slight. There is a little tenderness of the spine between the scapulæ ; some tenderness is complained of in the iliac fossæ, especially the left. Nothing is found wrong in the cardiac and respiratory systems.

As there was no doubt, from the history and circumstances, that the case was one of hysteria, a few days after admission I forcibly extended the left fingers, which had been rigidly flexed. In doing so the skin was torn at the roots of the fingers. A splint was then applied. Next morning, when the splint was removed, the patient was able to move her fingers to a considerable extent. The legs were now also forcibly moved at the knees, and the feet actively dorsal-flexed as far as was possible. There were evident adhesions in the ankle joints, which obstructed flexion. Some of these were broken down. Two days afterwards the patient could move all the joints of her legs a little. There was still aphonia. Although the grasp of the left hand still measured 0 on the dynamometer, she was found to be using the fingers

of this hand nimbly enough for knitting. After three weeks it is noted she has now very good movements in the left hand and arm, and has considerable grasping power with it. She can move the toes very well, and bend the knees considerably, but the feet are extended at the ankle joints and cannot be flexed on the legs. There is still aphonia, but she can close her glottis for the purpose of coughing.

About six weeks later the patient was discharged, able to walk fairly well, though a little awkwardly, without help. She could use her left hand and arm perfectly well. The voice had entirely returned. There was no rigidity of any of the limbs.

This was undoubtedly a case of hysterical paraplegia, and is important as illustrating, besides the incontinence of urine, some of the trophic disorders which may result simply from long disuse of limbs. The formation of adhesions in joints, requiring forcible disruption, I have seen in other cases which were proved by the sequel to be of purely hysterical origin. There was no real muscular contracture in this case, although the dropped position of the feet suggested overpulling of the sural muscles. As soon as the adhesions were all torn through, the feet could be dorsal-flexed into a normal position. I have reason to think that this condition of joints is often regarded as strong evidence against the diagnosis of an hysterical condition, but I have convinced myself that this is an error.

Nor was there any real muscular contracture in the left forearm, although the *primâ facie* resemblance to the late rigidity so often seen in hemiplegia, was remarkable. In true hemiplegic contracture it will be observed that you cannot by any amount of force straighten the whole limb at one moment. If you straighten out the fingers, the wrist remains rigidly flexed. Bring the metatarsus into a line with the forearm by extending the wrist, the fingers will *ipsô factô* become rigidly flexed. But in this case I found, as I have often seen in other cases of apparent contracture of hysterical origin, that I could extend the fingers and wrist at the same moment, thus bringing the forearm and hand and fingers into the same plane. In doing this, however, the

skin at the roots of the fingers gave way, so that there was a transverse tear at each metatarso-phalangeal joint, shewing that there had been an adapted contracture of the skin from long disuse of the member.

The preservation of the plantar reflexes in this case was an exception to the rule, which according to my experience is of almost universal application, that in hysterical paraplegia tickling the sole produces no response. Sometimes by persevering in a very elaborate titillation the reflex is produced, and occasionally also it is easy to see that a good deal of voluntary action is expended in restraining the muscular contraction. But usually there is a simple absence of the plantar reflex, the stimulus being felt as touch only, even in persons who are naturally very ticklish. The behaviour of the reflexes in hysterical paraplegia is almost always the converse of that which obtains in *tabes dorsalis*—the knee jerks are exaggerated and there may be ankle clonus—the plantar reflexes absent. The exaggeration of knee jerks occurs even when there is no tendency to spasticity in the limb, which, indeed, may be abnormally lax.

In tolerably recent cases of hysterical paraplegia the limbs may be firm and round, the skin healthy looking, and the knee jerks well marked. If in these circumstances inability to extend the knee joint and dorsal flex the foot is alleged, whilst the muscles respond normally to induced currents, and there is no spasticity, the diagnosis I take it, must be safe, because one can exclude the presence of lesion in and above the lumbar enlargement. But given a partial loss of power in a limb, with a tendency to rigidity, and the difficulty of diagnosis is often enormous.

Here we enter the field occupied by cases of incipient insular or disseminated sclerosis, and these are apt to offer diagnostic problems which are sometimes of almost insoluble intricacy. There are few diseases more easily recognised than disseminated sclerosis when typically developed; there is none, I think, which may present so much difficulty when the symptoms which combine to constitute the type are almost all absent, or but very faintly expressed, obscured too, as they continually are, by very evident signs of hysteria. I

was long ago indebted to an article by A. Pitres<sup>1</sup> for hints regarding the insidious modes in which this disease might make its approaches, but notwithstanding this and in spite of all the care which a peculiar interest in the subject has disposed me to take, I am conscious of having made not a few mistakes in diagnosis. From my own observation, and what I have known of the practice of others, I am convinced that a very large number of cases of an early stage of disseminated sclerosis—the stage in which there are probably patches of sub-acute interstitial myelitis—continue to be diagnosed either as examples of hysteria or of voluntary simulation. The most characteristic symptom of the disease is doubtless that tremor upon voluntary exertion which Charcot has so admirably pictured in his classical account of the disease. But this, as well as the articulatory difficulty, the rigidity of muscles, the nystagmus, and the so-called apoplectiform seizures, may be absent. The symptom which is perhaps least liable to fail is an excess of knee jerk, but even this may not be present—in rare cases, indeed, there may be no response to a blow upon the ligamentum patellæ—ankle clonus may not unfrequently be unattainable. Moreover, as is well known, exaggerated knee jerks are common in hysteria, and ankle clonus also is sometimes observed. The place properly (so to speak) occupied by the characteristic symptoms of the disease may be usurped by a temporary weakness of one limb, a transitory numbness in an extremity, loss of sight of one eye, a narrowly localized atrophy of a muscle group, or some so-called rheumatic pains. It is evident that when one of these symptoms (the atrophy alone perhaps excepted) stands by itself, and is obscured by very distinct signs of hysteria, the probability of an error in diagnosis is enormous. Indeed in the nature of things it is impossible in all cases to avoid this, but the chances of error are obviously diminished in proportion as we are alive to them.

I have been able in several instances to follow up cases which presented great difficulties to myself and others, and the sequel in these has proved so instructive that I make no

<sup>1</sup> "Anomalies de la sclérose en plaques."—*Revue Mensuelle*, 1877.

apology for detailing them. Unfortunately they are in some instances not quite so complete as would be desirable. Nor is it at all easy to describe with the pen the peculiarity of look and manner which so strongly suggest hysteria when one is in the presence of the patient.

A patient whom I saw on only one occasion nearly thirteen years ago, exercised my curiosity very much, and I should be very glad to learn for certain the ultimate issue.

A girl, aet. 20, was sent to me by her medical attendant on June 5th, 1877. There was a bad family history. Father epileptic. Several brothers and sisters died early.

Her mother, a very intelligent woman, had prepared the following notes of her case. "Almost total loss of the use of her legs, especially the left, which is very stiff. Formerly the right leg was most affected. Sharp pains through the right knee.

"Tingling feeling in fingers, with occasional numbness in the left hand.

"Mouth sometimes slightly drawn. At such times speech slightly affected. Appetite bad. Complains of feeling swollen. Her feet are generally cold. Her habit is costive. The breath is offensive.

"Her temper is peevish, variable, and hysterical.

"The urine is thick and high coloured : it reddens litmus paper and stains linen. Its specific gravity 1013. It contains no sugar or albumen. Perspiration is profuse and offensive."

The patient was a tolerably well-grown girl with red cheeks and large pupils. She could not stand without support, and then only with great difficulty. The legs, being bared, presented a helpless appearance—especially the left. The hair on the skin was rather long. The feet were cold and clammy. In trying to reach a chair in my room she fell down on the floor. I was told that she had fallen down several times at home.

In February, 1876 (about sixteen months previously), there had been a weakness of the right leg and the left hand, of which she got the better. Again, in the autumn of the same year, whilst at the seaside, there occurred suddenly

a weakness of the right leg. But just before she came to me the left leg became the weakest. She complained of tingling in both hands (most in the left), and said that she could not write, play the piano, or draw as she formerly could. She suffered very sharp sudden pains like a knife going through the right knee. There was also a dull aching pain in the left knee—especially in walking. A well-marked feeling of waist constriction was described.

Her articulation was peculiar—the words seeming to stick in her mouth. When at my request she held out a pencil in her right hand, and tried to measure the length of a face in a photograph, the arm shook up and down, so that she failed to do it.

In the left leg the patellar tendon reflex was strongly marked. When seated, if she drew the left foot towards her at a certain point spontaneous ankle clonus of a very marked kind was set up. This also happened when she lifted her foot from the ground and held it up. Tickling the sole also gave rise to clonus.

The heart and lungs were healthy. There was no spinal curvature nor tenderness on pressure. The ophthalmoscope showed no change. There was no nystagmus. In childhood she had been rickety, it was said, and she did not walk until more than three years old.

Her mother told me that within the last year or two she had become completely changed. She had taken strongly to ritualism. Her moral character had degraded—the girl having become peevish, ungrateful, and strongly inclined to prevarication. It appeared to me that along with the hysterical symptoms there was typical evidence of disseminated sclerosis, and I expressed a distinctly unfavourable opinion as regarded the future. Arsenic was suggested, and a visit to the seaside.

A few months later I learned that she had quite recovered. But I could never get the opportunity of seeing her. In July, 1878—a year after she had been brought to me—I learned from her medical attendant, who frequently saw her socially (not professionally), that the girl seemed quite well. She could walk any distance, occupied herself, and appeared,



as far as he could judge, to have nothing whatever the matter with her.

In answer to an inquiry of mine a year and-a-half after this (two and a-half years from the date of my examination), he wrote:—"When I last saw Miss T. she was very well, but she left E—— about three months ago, and has gone to reside in D——. From time to time she has limped a little, or squinted, and then has taken the arsenic. I cannot help thinking her symptoms are due to that wonderful simulator hysteria."

Since that time I have been able to obtain no information about this patient. I have still no doubt that it is a case of disseminated sclerosis, but the narrative illustrates some of the difficulties encountered in coming to a conclusion as to the functional or organic nature of certain symptoms.

The fact that in the course of disseminated sclerosis there are apt to be periods varying in length, of almost or even complete subsidence of symptoms, though mentioned by Charcot and writers generally, is not yet, I think, sufficiently remembered and taken into account when we are occupied with the diagnosis of a case which presents difficulties. The circumstance of this girl's apparent recovery would not unnaturally lead to the conclusion that the case had been simply one of hysteria, which the consultant had mistaken, but it will have been observed that a year after the supposed recovery mention is made of occasional "limping" and "squinting," expressions which in the circumstances are of grave significance.

Before remarking on the value of some individual symptoms, I should like to record some cases which originally gave rise to a difficulty of diagnosis which has been resolved by the sequel.<sup>1</sup>

A lady, æt. thirty-one, was brought to me on February 7th, 1878, on account of loss of power in the lower extremities. She had been quite well, I was told, though never very robust, until two years previously, when she

<sup>1</sup> Many of the cases which follow could not be read at the meeting for want of time.



began to complain of numbness in the feet, and shortly afterwards in the right hand, and also of stiffness in her joints. There was also some numbness in the left side of the face and head for a short time.

At first she could not feel the ground with her feet, and then by degrees there was failure of power, so that she tottered in walking. This had gradually increased, and at the time of her visit to me she could not apparently walk across the room without taking hold of the furniture in order to balance herself.

Now and then she had a feeling of waist constriction. Sometimes there was delay in passing urine. At other times she could not hold it. The bowels were always confined. The grasp of the right hand was almost *nil*. In picking up small things from the table there were rhythmical movements of the hands. The faradaic excitability of the arms was normal. She complained of pain, or a dull weight at the back of the head; there was no noise in the ears. As she sat everything seemed going round. She had had no vomiting. Always myopic, her sight had latterly become feeble. With all this she had rather gained flesh.

With the ophthalmoscope I found the right disc more pale than the left, which, for its part, was of a dull grey hue. There was no posterior staphyloma. She could see much better with the left than with the right eye. There was no nystagmus. She had a sallow aspect. The heart and lungs were healthy. Three sisters had died of consumption.

On the right side there was ankle clonus. When she pointed the foot it was moved wildly to and fro. The patient was sent to me as a case of hysteria.

It was clearly an example of disseminated sclerosis. I did not see her again, but learned a year or more afterwards that she had become decidedly worse.

I saw, November 23rd, 1881, with Dr. Playfair, a young lady, twenty-three years of age, who could not walk without help, and the question to be determined was whether the case was one of hysterical paralysis, which it had been pronounced to be by many of the medical men who had been consulted. Dr. Playfair was inclined to think that it was

not a case of hysteria. The patient who was unwholesomely fat lay in bed. I was struck at once with the fact that she had a little horizontal nystagmus; the pupils were moderate in size; she described herself as being short-sighted. Some five years previously she had complained of her eyes, and had been advised to go to the seaside.

In 1876 she strained her left knee, and was lame in that leg for two or three months afterwards. In 1878 she had a fall on the back, and since then she had gradually lost power in both legs, in the left especially. There was no loss of sensibility. I found the knee jerks excessive in each leg and ankle clonus in the left, rather strongly marked, slighter clonus in the right. The reflex from the soles of the feet was very imperfect and suggested a hysterical condition. The legs seemed flabby and the skin was coarse and cold and inclined to the purplish tinge that one often sees in the legs of hysterical patients.

As she lay in bed with her hands upon the bedclothes in repose they were quite quiet, but when at my request she put out her right hand to pluck a grape from a bunch, the arm immediately betrayed the rhythmical movements characteristic of disseminated sclerosis in a marked form. The same symptom was noted though to a less extent in the left arm; so also, when she sat up in bed with help, the muscles of the trunk became affected with movements and the head began to shake. When put upon her feet the muscles of the trunk and extremities felt rather rigid.

I examined the eyes with the ophthalmoscope; the right disc showed no important change, but the left presented that dark grey discoloration (resembling the tint of hyperæmic grey matter of cerebrum) which I have seen now in several cases of disseminated sclerosis. My opinion was that the patient was affected with disseminated sclerosis. Dr. Playfair recently informed me that this young lady was brought back to him six years afterwards in the hope that he would undertake her case which, however, he declined. By that time the symptoms of disseminated sclerosis were very marked and far advanced.

The following case is extremely interesting. Mrs. ——

was seen by me in consultation with Dr. Playfair on March 13th, 1883. She was the mother of four children, the last child having been born a year previously. She had been ailing for two or three years, and complained of loss of power in the lower extremities. There was partial rigidity of the legs, which were not ill-nourished. The soles of the feet were very ticklish and the legs moved up vigorously when they were touched. Tendon reflex was in excess and there was ankle clonus. As she lay she was unable to move either foot from off the bed. She complained of a sense of tightness around the ribs. Examined by the ophthalmoscope the right optic disc was seen to be white and presenting that metallic glistening look which is observed in some states of atrophy.

There was a history of loss of sight of the left eye with gradual recovery, and later, of loss of sight of the right eye with gradual recovery, which had been thought to point strongly to hysteria. In spite of this, I believed from the symptoms that the case was probably one of disseminated sclerosis. She was afterwards seen by a distinguished colleague, who thought that the condition was hysterical, and she went under Weir Mitchell treatment in the hands of Dr. Playfair.

I learned only a few days ago from a friend of this lady that she "sometimes walks five or six miles to the services of her church on Sundays." Her husband's report of the same date is "Partial only, and I fear never likely to be complete recovery. Greatly improved power of locomotion, less nervousness, but a great deal remaining, and decided inability to think of much at any time."

A lady, aged 27, the mother of two children, the last being three years old, was seen by me in consultation with Dr. Playfair, on June 6th, 1883.

The patient who said that she had enjoyed good health till two years previously, did not present the aspect of illness in her face. Her complaint was of difficulties in walking and also in the sense of sight. When examined I found that she could not guide her steps rightly, and her head shook with every step that she took. She could not go through a

door straight. If she ventured to look back in walking she would fall down from a feeling of extreme giddiness. She had been numbed all over her body. The left leg appeared to her to be weaker than the right, the knee jerks were present and well marked in each leg, more in the left than in the right. There was no ankle clonus. No tremors of the limbs were noted. The wrist jerks were well marked. She did not suffer from headache, or deafness, and there was no facial paralysis. The patient's manner was highly hysterical.

It appears that she lost the sight of one eye (the left) entirely two years ago. It recovered to a certain extent in about six weeks. One year ago the sight failed in the right eye, though not to the same extent as it had done in the left. At the time of my examination she could not read or do needlework after exerting herself; she was always obliged to lie down first. After resting thus for a few minutes she was able to read a book. She read No. 2 type at four inches; the right pupil contracted to light; I was unable to be certain about the contraction of the left. Her appetite was bad: she did not suffer from any pain, but was depressed in spirits. She had lost all numbness, except in the tip of the tongue and the fingers. It appeared that two years previously she had felt numb all over the body; the numbness began in her waist, and descended to the feet, and then mounted from the waist to the throat. The ophthalmoscopic examination showed no change in the optic discs. As this patient quitted the house walking, I noticed her gait, which was staggering like that of the intoxicated.

She appeared to be very quick and intelligent, and this her husband confirmed. He said, however, that she was emotional and inclined to be sentimental. He told me that two very distinguished authorities had both considered the condition hysterical.

I wrote to Dr. Playfair that I could detect no distinct evidence of organic disease.

It seems that when the eyes failed she was taken to an ophthalmic surgeon, who examined her and found nothing perceptibly wrong. He sent her to an eminent colleague, who took a grave view of her case. This patient was sub-

mitted to systematic treatment by Dr. Playfair, who, in answer to my enquiry, writes as follows:—"I treated this case. I have a note, July 21st, 1883, to this effect: Mrs. J— got quite well, and could walk with perfect ease, but she will not take my advice about going away with her nurse, and is going straight home, and I fear she will relapse. This fear appears to have been justified, for I have a letter from her husband, dated September 24th, 1883, in which he says: 'At first my wife was able to walk a mile or a mile-and-a-half, but she did not seem to make further progress, and latterly she has lost ground very much, and her sight is worse than before, and she can now only walk a very short distance.'" For my part I should have little doubt from the last paragraph that the case is one of disseminated sclerosis, but I have failed to obtain any more recent information regarding her.

A lady was seen by me in consultation with Dr. Playfair, November 27th, 1884. She was very deaf, fifty-three years of age. I found her lying in bed; she could not completely flex the knee joint, though Dr. Playfair had seen her do it on the preceding day. In trying to do this she lifted the whole lower extremity off the bed. The right limb was alone affected, the foot was flexed in the plantar position, apparently from semi-contraction, whether or not of the plantar fascia I am doubtful, but certainly there was contraction in the sural muscles. As she thus lay in bed, the tendo achillis was felt to be very rigid, but when I got her out of bed, seated, with her foot on the floor, this rigidity was not noticeable whilst she sat with the leg at right angles to thigh. In this position, unless by a determined effort (looking meanwhile at her feet), she brought the heel down, this part of the foot remained off the ground, but apparently she could put the flat of the foot on the ground. It was, therefore, impossible to say whether or not there was any permanent contraction of the sural muscles. With a view of helping towards the solution of this point I tested the anterior tibial muscles of the right leg, which responded normally (considering that they were smaller than the left) to induced currents.

The knee jerk was exaggerated on both sides, and there was some ankle clonus in the right leg. There was no affection of the bladder or bowel.

Her symptoms dated from two or three years back. Several years previously she had lost the use of the left arm and leg for a time, but recovered on going to Switzerland.

There was a sense of numbness in the right leg, as if it were covered (as she described it) with an extra skin of kid. The plantar reflex was good in both feet. Frequently she did not know where her leg was, and when she got out of bed it seemed quite useless and an encumbrance. It tended to wave about in a "drum-stick" fashion. She described her leg as being better than it had been, and she said that both her leg and thigh were bigger than they had been. She complained of a soreness in the left sacral region, which she felt especially on putting the right foot down.

The opinion was given that the condition was of functional character, and she was put under systematic treatment by Dr. Playfair. He reports to me, Nov., 1889: "This patient got apparently quite well. When she left me she had got rid of her crutches, sticks, &c., and could walk about. I have not since heard of her, but her nurse tells me she heard about a year ago that she was fairly well, but still could not walk perfectly." This would be four years after treatment.

The sequel as thus described, as well as subsequent experience in cases presenting the symptoms noted, make me think that I was wrong in my opinion, and that the case was one of organic disease, probably of disseminated sclerosis, which had evidently, however, derived great benefit from the treatment. I would note here that I was probably unduly influenced by the statement that several years previously she had lost the use of the *left* arm and leg for a time, but recovered on going to Switzerland. I shall have to say something more on this point later on.

A well-grown, healthy-looking lady, æt. twenty-five, of good figure and development, was seen by me in consultation with Dr. Playfair on Nov. 17th, 1886.

Until five years ago—*i.e.*, when she was twenty—her

health had been good. She was very active, particularly fond of walking, and able to walk nine miles. Then, without ascertained cause, she began to get some powerlessness of the left leg, which was at first thought to be rheumatic. It went on increasing so that ere long she could only walk half a mile. Since that time, although she had varied apparently a good deal—being sometimes scarcely able to move her legs and at others walking pretty well—she had never been able to walk two or three miles. The loss of power, which began in the left leg, shortly extended to the left arm, and afterwards the right leg and right arm became affected. It would sometimes be the right, sometimes the left lower extremity which was most weak, and so likewise with the arms. Moreover it would occasionally be the leg of one side and the arm of the other which would be co-incidentally affected.

The patient walked in a tottering way. The muscles flexing the thigh appeared to be stronger on the left than on the right side. She could get up on to a chair with either leg first, but with the left better than with the right. The muscles appeared to be well nourished. Her power has varied very much from time to time. Five years ago she had a manifest squint with double vision, which lasted for several months. It was on account of the squint that she was taken to an eminent physician, who expressed an unfavourable opinion of the case. The knee jerks were exaggerated, and ankle clonus was said to have occurred, but I did not elicit any. An opinion was given that the condition as then observed was of functional character, and systematic treatment advised. Dr. Playfair treated her, and she apparently got so far well, that when treatment was discontinued there was only a slight difficulty in walking. From recent enquiry, however, it appears that now, three years later—she remains *in statu quo*, and is evidently affected with organic disease.

A lady, aged thirty-nine, the mother of one child, was sent to me by Dr. Playfair on July 9th, 1888. She complained of loss of power in walking. In Scotland, four years previously, she could walk six miles, and gradually after that



she lost power, staggered in her walk on looking up, and was obliged to hold on to something. At the same time, she said, a touch was not felt on the left side of the forehead and cheek, and the mouth was pulled on one side—she thinks the left. When examined, it is noted that her hand is steady for holding or writing; the ends of her fingers tingle. Her speech is of a hesitating clipping character, but this, it seems, she has had since the age of fourteen. Her sight began to fail in 1882; the failure in her walking powers a year or two later. The knee jerks are well marked; there is no ankle clonus. She picks up anything from the table easily and without tremor. Three years ago she could not see to read and write, but recovered the power, and for two years she could read fairly small print. Now she can hardly read minion (No. 4) at all. Both optic discs are found to be distinctly atrophic.

Dr. Playfair informs me that this patient apparently made a good recovery under systematic treatment, and went abroad with her nurse. For two and a-half months her condition remained quite satisfactory. Then one day after unusual fatigue she had a cerebral attack, the precise nature of which is left in doubt. She appears to have been quite unconscious for a considerable time, perhaps some days, and was supposed to be dying. Eventually she recovered from this and came back to England.

I saw this patient on May 29th, 1889, and examination shewed distinct symptoms of disseminated sclerosis. The cerebral attack was probably of the apoplectiform character described by Charcot as occurring in cases of disseminated sclerosis.

A lady, æt. twenty-four, was seen by me with Dr. Playfair, on May 14th, 1884, when the following notes were made. She has a good colour, large pupils, and is not unhealthy looking. She is rather, but not very thin. She rises from a chair with difficulty and stands tottering, glad to put her hand on anything to support her, and elects to have the feet wide apart, so as to enlarge the base. In walking, the gait is somewhat ataxic, with a slight tendency to over-action. There is but little power in the flexors of the thighs. The



knee jerk is much in excess on both sides, and there is ankle clonus. The plantar reflex is present and not abnormal. Complaint is made of considerable anæsthesia of the lower extremities. A touch is felt, but dimly and less distinctly on the right than on the left leg, and she cannot distinguish by the touch of her foot a Turkey from other carpet. In reply to a suggestion, she says that her legs feel as if asleep. She does not know where they are in bed without kicking against something. There is slight cutaneous anæsthesia in the fingers. She cannot pick up small things without looking. She has had cutaneous anæsthesia of the left side of the face, and indeed more or less all over her body. No bladder trouble is reported, except rather frequent micturition. There is no nystagmus.

The patient's illness dates from three years. First there was deafness, or rather sounds were heard as though at a great distance. Then followed diplopia; there was no strabismus perceptible, according to her sister's account. A surgeon said there was paralysis of a nerve, but an ophthalmic authority is reported to have said that it was of no consequence. In a week or two or more she lost the sight of the left eye almost entirely, but after some little time it returned. Now nothing abnormal is to be detected with the ophthalmoscope. She says that now the sight of the *right* eye is not so keen as that of the left.

It seems that at one time she had lost the use of both her arms. Now there is good grasp with each hand; no increased reflex is to be observed at the wrists. There are no tremors in the arms.

An opinion had been given by two eminent physicians three years previously that this patient had disease of the spinal cord, but two equally distinguished authorities who had more recently seen the case (one of whom had indeed sent the patient to Dr. Playfair) said that it was an example of hysterical paralysis.

Having been informed that three years previously the anterior muscles of the left leg had been found by a high authority to give no response to induced currents, whilst those of the right contracted well, I submitted the patient to a careful examination with electrical currents.

The application of currents to the legs gave rise to a good deal of reflex muscular action in the muscles moving the thighs. The Faradaic excitability was everywhere quite normal in the lower extremities, as also in the forearms and hands. With the galvanic current K S Z > A S Z. I gave the opinion that all the symptoms observed at that time could be explained by functional trouble, but some reserve must be felt owing to the past history.

The patient underwent a course of systematic treatment for nerve prostration, but without satisfactory result as regards her power of walking.

At her mother's request I saw her nearly four years afterwards, February 27th, 1888. She was then a good deal more helpless, but could lift the right knee a few inches, and the left, perhaps, one inch. She could not dorsal-flex either foot. With a stick and an arm she walked in a laboured manner, and the exertion to do this caused her great fatigue. Her eyes now presented nystagmus, both horizontal and vertical. There was a little waviness in the movement of the hands. Speech and hearing were good. Patient suffered from great frequency of micturition, every quarter-of-an-hour needing relief. She had no trouble with the bowel. She slept well, and her general health was good.

Tested by induced currents the anterior tibial muscles of the left leg shewed more defective response than those of the right, but on both sides the reaction was distinctly lowered. It does not need to be said that symptoms of disseminated sclerosis were now strongly pronounced.

In the case which follows a diagnosis of hysteria had been made before it came under my observation, and the girl certainly presented very much the aspect of an hysterical patient. It seems worth recording because of the sequel.

A young woman, æt. twenty-two, was admitted into the National Hospital for the Paralysed and the Epileptic on August 3rd, 1887, on account of loss of power in the hands and also in the lower extremities. There was no history of injury. She had suffered from measles when a child. About five years previous to admission she began to suffer from pains in both hip joints and weakness in the legs. Nine

months ago her knees gave way, and she complained of severe pains in the lower limbs. For three months she was able to go about with the aid of sticks, but for the last four months had been confined to bed.

For the last three years she had had attacks of loss of power in the hands, accompanied by semi-flexion of the fingers and pains.

On admission it was found that she could perform all movements of the upper limbs. No wasting or rigidity was present. She stated that she had occasional numbness and a pins-and-needles sensation in the fingers of both hands, and that she often had loss of power in the hand for a short time. No loss of sensation was observed. When asked to take hold of a pen held at a distance, there was marked tremor and inco-ordination. This was present in both upper limbs. It was with difficulty that she could drink from a cup without spilling the contents. The wrist jerks were present on both sides, not exaggerated.

She could perform all movements of the right lower limb as she lay in bed, but they were feeble. The left lower limb was flexed at the hip and knee, and patient was unable to extend fully at these joints. She was able to dorsal-flex the foot to a slight extent, also to flex and extend to a very slight degree the left hip and knee, but was unable to perform any other movements of the left lower limb. There was drooping of the toes of both feet, especially the left foot, but the patient was able to extend them. There was great rigidity in both lower limbs, especially in the left, which resisted all attempts at passive movements. The plantar reflexes were present on both sides, ankle clonus present on both sides, especially marked on the left. The knee jerks were exaggerated; there was knee clonus on the right side.

The right lower limb was subject to attacks of great rigidity in a position of extension; the left to attacks of rigidity in a flexed position. Formerly, according to her account, the left limb was apt to fall into a rigid posture in the position of extension.

There was no loss of sensation anywhere, but the patient complained that she suffered from twitchings and drawing

up of the legs, especially of the left. She also had pain in the thighs and knees. She had been unable to walk for the last three months.

When examined she was unable to stand without help, and she could not put the left foot to the ground on account of the contraction described. There was slight nystagmus and tremor of the head and neck when the patient was asked to fix her eyes on any object. Her speech was rather slow, and there was a tendency to pronounce the words in syllables; there was very slight tremor in the lips and tongue. She suffered occasionally from pain in the head, in the vertex, and also in the forehead. She described having frequently suffered from diplopia. The pupils reacted to light and to accommodation.

There was no spinal deformity and no tender spot on percussion of the spine. There was no vesical trouble, and nothing wrong in the respiratory and circulatory systems.

The optic discs were very pale. On sustained convergence there was well marked quick nystagmus occurring simultaneously in both eyes, but generally better marked in one than in the other. The pupils were not observed to vary consentaneously with the nystagmus.

The patient having been attacked with scarlet fever, was sent to the Fever Hospital, where she died. Dr. Barlow, under whose care she was admitted, was good enough to let me examine a hardened specimen of the spinal cord, which showed characteristic evidence of disseminated sclerosis.

During the first week or two of the following patient's stay in Hospital, I could not make up my mind as to the nature of her case, and I distinctly inclined to the belief that it was of functional character. She was highly hysterical; there was a history of long continued vomiting, and of loss of voice. But further observation convinced me that it was one of disseminated sclerosis—as was proved by the sequel.

A female patient, æt. forty-two, was admitted into the National Hospital for the Paralysed and the Epileptic, Queen Square, on Dec. 3rd, 1884, complaining of stiffness of both legs, being unable to walk without aid; also of pain, especially in the back, which seemed to radiate down both legs,

shooting in character. The left leg appeared worse than the right. The pains were better when the patient was up and walking about.

For the last twenty years the patient had been subject to attacks of sickness after every meal (sometimes only after one meal in the day), the longest interval of freedom from sickness at any time being 14 days. During this time the patient used to be readily tired, the legs would ache, but she did not have any special pains.

Four and a half years ago the aching and weariness of the limbs began to get worse, and the patient complained of the weight of her dress. The calves of both legs used to swell, and become shiny and hard. This disappeared in about a fortnight or so, but would occasionally recur. After this she began to suffer much from sharp shooting pains in the head and neck, relieved by lying down. The toes also at that time became very tender, and the tenderness continued for about two months, but had not returned.

Twelve months ago the patient noticed the legs begin to get stiff, but even before this her legs had occasionally drawn up when she was in bed. On two occasions during the last fifteen months, the urine has had to be drawn off.

It is noted on admission that the patient is fairly well nourished. The hands tend to turn over, and she would drop things unless very careful; the grasp is fairly good. She is not able to do fine needlework. Occasionally there is a pins-and-needles sensation in all the fingers of the right hand. She has difficulty in touching the nose with the fingers, her eyes being closed, but there are no tremors. She is able to localise touch and pain correctly in the arms; the wrist jerks are not increased. There is no nystagmus.

She has a feeling of tightness around the waist; the whole leg from the knee to the ankle feels tight as if it were going to burst. Tactile and painful sensibility is much impaired all over the legs and thighs. The plantar reflex is well marked in both legs; both legs are very stiff. Knee jerk is well marked when the legs are not too rigid, but it is impossible to get ankle clonus because of the stiffness of the legs. She is able to direct each foot pretty well to an object.

As regards the sickness from which she used to suffer, she says that she never felt sick, but food would come up at once. There was no pain in her stomach; she was obliged to get up from the table; the smell of fish would make her sick. Eight years ago for about three months she lost her voice. Her manner is highly hysterical.

This patient died in 1885 in Guy's Hospital under the care of the late Dr. Carrington. The notes of the autopsy, for which I am indebted to Dr. Gay, are as follows:—Large bed sores over the sacrum extending down to the bone, and one over the great trochanter of the left side. Abscess in each thigh, which had been opened. Brain weighed forty-one ounces. Meninges and arteries at the base presented nothing abnormal. The left temporo-sphenoidal lobe shewed yellow softening on the surface and interior extending to the corpus striatum. The whole brain was very carefully cut into fine sections, but no sclerotic patches were found. The spinal column was normal. A patch of purulent lymph was found on the posterior surface of the spinal cord in the lumbar region. The cerebro-spinal fluid was rather in excess. Patches of sclerosis began in the pons varolii, at the centre of which there was a patch on either side, and one also on the surface. They are raised, hard, semitranslucent and glistening. There is a patch on the surface of the medulla oblongata, spindle shaped, half-an-inch long and extending inwards to the white matter. There is no softening of the cord but many patches of sclerosis on the surface or more deeply.

The dorsal region of the cord was the least affected, the lumbar most. The posterior columns were most diseased, many parts between the posterior cornua and commissure being completely sclerosed. In some parts of the lumbar enlargement the whole transverse area, including the grey matter, was affected. All the other organs were healthy.

A case which was quite recently under my care in hospital furnishes so good an illustration of the difficulty in diagnosis that may be caused where a moral shock forms part of the history that I think it worth recording.

Ann B——, æt. thirty-one, widow, was admitted into

the National Hospital for the Paralysed and the Epileptic, under my care on October 26th, 1889. The following notes were taken by Dr. Taylor, resident medical officer.

Nine years ago, two or three weeks after the death of her husband (after a fortnight's illness), which had been a very great shock to her, she suddenly in one night lost power in her legs, and was unable to stand or walk for several weeks. She gradually recovered, but has never been as she was before—has never been able to run or to walk fast.

During the last six months she has gradually lost ground, not suddenly, but she is now worse than she has been during the last seven years. Her difficulty is to get her legs to separate from each other, and also in going up stairs. For the last four years there has been at times great difficulty in passing water, and sometimes she has suffered from incontinence. Patient was a strong healthy woman previous to her husband's death. On admission she complained of difficulty in walking. The condition of the arms was normal, except that a slight tremor showed itself at the end of the act in attempting to touch an object with the left hand. In the lower extremities all movements were carried out though rather feebly and with some tremulousness, especially to be noted on bending the knees.

There was a peculiar condition of the skin, of ichthyotic character, which appeared to be sufficient to account for any blunting of sensibility observed. This has been present according to the patient's account, all her life. The knee jerks were greatly exaggerated. Ankle clonus was present in the right leg, and a tendency to it was evident in the left. The patient appeared well nourished. The gait was that of a person with functional paraplegia, but the legs shewed notable stiffness. When standing the legs were widely separated and became tremulous, whilst the body swayed to and fro.

Under observation in hospital this patient has continued much in the same state. There is no peculiarity of speech and no nystagmus. The ophthalmoscope shows no change in the optic discs.

I am disposed to think that this woman is affected with



organic disease of the spinal cord, and probably in the form of disseminated sclerosis, but the difficulty of pronouncing an opinion will, I think, be manifest.

I am conscious that these records do not serve to present the consultant, whose diagnosis has been exposed to the criticism of time and treatment, in a particularly favourable light. That is a misfortune for me, but not, I trust, for my audience. And I would venture, in a very modest spirit of self-defence, to say that these cases have been selected for the reason that in many instances the sequel was not that which had been expected—with the express object indeed of showing in what the difficulties of diagnosis may consist. It would have been easy enough to have brought together numerous instances in which the result proved the accuracy of the diagnosis, but that would have been little to the purpose of my address.

There can be but little doubt that of all organic diseases of the nervous system, disseminated sclerosis in its early stages is that which is most commonly mistaken for hysteria. This is evidently due especially to the following circumstances. The disease is particularly common in young females—symptoms showing themselves about the period of puberty. There is very often a history of some moral shock immediately preceding the first symptoms. It is a question well worthy of consideration whether sudden strong emotion or long continued mental worry may not be found to be important etiological factors in the disease, but this is not the occasion for discussion of the point. In addition there are few cases of disseminated sclerosis in females in which marked hysterical symptoms are not mixed up with those belonging essentially to the disease. Obviously this combination of itself causes a peculiar liability to mistakes of diagnosis. But there are other sources of error in the fact that many of the symptoms of disseminated sclerosis are supposed to suggest of themselves an hysterical origin. A sudden alleged loss of power in a limb of an apparently healthy young female, a localised numbness, or pins-and-needles sensation, complaint of loss of sight in one eye, are symptoms familiar enough as expressions of functional



trouble. They represent equally modes in which organic disease of the kind we are discussing may make its first appearance. These local symptoms may clear off after a short time, just as would be the case if they were of hysterical origin. The girl recovers her sight, or the use of her limb, and nothing more is heard of the numbness. A little later perhaps loss of sight in the other eye is complained of; a pins-and-needles sensation is described in some other part; another limb is said to be very weak. The opinion that the symptoms are of hysterical origin may very possibly appear to be absolutely confirmed by this reappearance of trouble in other situations. Or the patient perhaps complains of weakness and stiffness in both legs, which increase so that in six or eight weeks she cannot stand. Then comes a rather rapid improvement and she recovers her power completely, soon, however, to fail again. After recoveries and relapses of this kind, the characteristics of confirmed disseminated sclerosis show themselves.

In the other sex also in persons of neurotic temperament and inheritance there may be a great liability to error. I will give an instance.

In 1882 I examined in consultation a young man with sallow complexion, large pupils, and very nervous manner. He was lying on a couch, and said that he was not able to walk much, because walking brought on a queer sensation in his legs, especially in the left leg, a pins-and-needles numbness being described. There did not appear to be any anæsthesia of the legs. His symptoms were entirely subjective. It appeared that at Easter, 1881, he played fives during a very cold wind. He was dressed in flannels, but felt it cold to his legs, and thought that he would get a bad chill. Later there was a feeling of numbness, especially in the left leg. Since then, with intervals of good health, he had had several breakdowns, which had occurred after walking too long a distance, jumping, riding, some physical strain. There had been no moral strain.

I tested the muscular power, which seemed perfect in all the groups of muscles of both lower extremities. The knee jerk on each side was much exaggerated, and there was a

slight attempt at ankle clonus, more on the right than the left side. There was, however, considerable excess of reflex in the triceps and wrist also, whilst there was no complaint of anything being wrong with the arms, so that I thought the excess in the knees might be "discounted." There were no tremors. Plantar reflex was not very strong.

The pupils contracted readily to light; the ophthalmoscope showed nothing abnormal. His tongue was covered with a thick fur, probably from the bromide and iodide which he was taking. There was a strong neurotic history on the mother's side. I diagnosed a functional disorder, and advised that the patient should go for a voyage and then resume his studies.

I saw this patient about fifteen months later. It appeared that after seeing me he went for a time to the seaside, and thence to one of the Universities, where he took his degree satisfactorily, and appeared to be quite well. Soon afterwards he walked in Scotland as well as ever. He only complained of a little numbness about the knees, equally on each side. I congratulated myself on a successful diagnosis, but this, as the result showed, was premature. Not to weary you with details, it is enough to say that this patient now presents characteristic symptoms of disseminated sclerosis.

In view of this communication I have looked through a large number of notes of cases both of hysterical paralysis and of disseminated sclerosis which have been under my care, and in many of which I have been able to learn the issue. As regards the value of individual symptoms in enabling us to differentiate organic disease from functional disorder, I find that whilst there is still much to be desired, there are points in which experience appears to speak with a fair amount of distinctness.

As a rule, though this is not without some notable exceptions, the class of hysterical paraplegia is not difficult of diagnosis by those well acquainted with the symptoms and course of organic disease, the surrounding circumstances, and especially the contradictions palpable in the symptoms leaving one usually in but little doubt. I need not dwell upon these before my present audience, but would remark

that the attitude and conditions of the lower limbs may vary exceedingly. The limbs are most often in a state of perfect flaccidity, a condition of spasticity being comparatively rare. The feet are frequently "dropped." After long disuse it will not unfrequently happen that there are strong adhesions in the joints. I have already referred to this, and to the pseudo-contracture due partly to this fibrous ankylosis and partly to contracture of the skin—not of the muscles. Hysterical paralyses are most often complete. The loss of power in disseminated sclerosis is very rarely (except in advanced stages) more than moderate. I cannot help thinking that the view still generally held that a shifting of loss of power from one limb to another (such as that which I have described) is really characteristic of hysteria is quite an error. The hysterical woman who has lost all power in her legs, will, it is true, very often later on (whilst still paraplegic) lose the power of one arm, usually the left; but I have not found that she is prone to lose the power in a limb, then recover it, and then lose it in another. It seems to me that the idea of this shifting of powerlessness being strongly suggestive of hysteria has arisen from the mistakes in diagnosing as hysteria cases of disseminated sclerosis, which must have been continually occurring before the latter disease had been differentiated. No doubt the hysterical are prone to changes of disorder; at one time, for example, losing the use of a limb or limbs, with or without profound anæsthesia, at another losing the voice, or closing one eyelid, or contracting a limb, but the shifting about of a state of more or less powerlessness, which we see in disseminated sclerosis, appears to me to be *sui generis*, and should, I am disposed to think, save us from error. And equally so with the occurrence of numbness or pins-and-needles sensation, sometimes at one part and sometimes at another, which, if my notes do not betray me, points with considerable distinctness to disseminated sclerosis.

There would appear to be a little more difficulty in regard to the impairment of sight in one eye to which I have referred. The ophthalmoscope perhaps shows no change. But we shall find, I think, that the hysterical patient as a

rule, when loss of sight of one eye is in question, is quite blind on that side, whilst the patient with sclerosis has only more or less obscurity of vision. I cannot call to mind, since I have been better acquainted with disseminated sclerosis, any case of simple hysteria in which first one eye lost some amount of vision for a time and recovered, and afterwards the other eye behaved in a similar fashion. So that this symptom I should now take to point with considerable force to disseminated sclerosis. When the ophthalmoscope shows atrophy of disc (and it is remarkable in what a large proportion of cases of disseminated sclerosis some atrophy is to be found—in some a stage of hyperæmia preceding it) my experience would teach me that a diagnosis of functional disorder must be discarded.

The same must be said of nystagmus, a symptom of peculiar value when combined with others about which there might otherwise be some doubt. It is necessary, of course, to remember the possibility of chronic alcoholism producing a temporary nystagmus, but this chance of error ought not to be difficult to avoid.

We next come to the tremor on intentional movement, upon which I am disposed to place a diagnostic value higher than that possessed by any other symptom of disseminated sclerosis. Looking back many years I can remember observing numerous cases which presented this symptom at a time when I used to feel very great difficulty in the differential diagnosis which we are considering. I cannot call to mind one which the sequel proved was simply functional. It is true that in the hysterical we not unfrequently see a clumsiness of movement of the hand when directed towards an object, which is somewhat liable to deceive, but observed carefully it will be found that this is rather of the nature of ataxy than a rhythmical tremor such as is found in sclerosis.

There is also another variety which is worth noting. The patient asked to touch an object with her finger, does so without difficulty or hesitation, but when the finger has rested upon the object for two or three seconds the arm becomes affected with somewhat rude tremors. This is in striking contrast with the tremor which affects the arm in disseminated

sclerosis, as the patient brings the finger near the object, tending to cease when it is attained.

There may be very fine and rapid tremor only when the patient stands upon the feet, ceasing when the sitting posture has been assumed.

On the coarser semi-convulsive movements, twitchings, jerkings, and grimacings not rarely met with in hysteria I do not dwell. They could not for a moment deceive any one acquainted with the subject.

Localised atrophy of muscles with loss of electrical reaction is well known to occur sometimes in the course of disseminated sclerosis, and in a case otherwise open to doubt its presence is undoubtedly of the highest value in determining the organic nature of the disease. But I do not think it is generally known that the localised atrophy may behave like the temporary powerlessness of a limb or limbs, or the shifting numbness. I have seen several cases of disseminated sclerosis in which atrophy of some muscles, with loss of electrical reaction, has cleared off entirely, to be succeeded some time afterwards by a similar lesion in another or the same part.

It is scarcely probable that disseminated sclerosis is a new disease. Little more than half a century has elapsed since it was first figured by Cruveilhier in his "*Atlas d'Anatomie Pathologique*," and twenty-two years ago Charcot expressed his belief—a well founded belief, I have reason to think—that the disease was not known, that is to say not recognised, in England. It is practically indeed to Charcot that we owe our acquaintance with the disease, from the admirable summary of its clinical and pathological features published by him in his earlier lectures. We all know what a length of time it takes for a disease, however excellently pictured, to fix its features so firmly in the minds of medical men generally as to make the diagnosis of it come readily to those who have not gone out of their way to seek examples of it. And this, which is true of most forms of organic disease, is from the nature of things most marked in reference to disseminated sclerosis. In that disease the infinite irregularity in the situation of the essential pathological

lesions creates difficulties of recognition beyond those to be met with in any other example. This being the case it is not surprising that the symptoms, characterised as they are by frequent remissions, should, in the absence of other explanation, be set down to the vagaries of hysteria. The almost constant admixture of circumstances pointing to an emotional origin or accompaniment of these symptoms greatly increases the likelihood of this confusion occurring. As I have before remarked, it appears to me reasonable to conclude that many symptoms which have come to be considered characteristic of hysteria will, if examined by the light of improved knowledge and experience, be relegated to disseminated sclerosis. The same principle holds good, as I have already shown, in reference to atrophy of the iliaco-psoas muscles, and to Friedreich's paralysis, not to mention others. But in none so much for the reasons mentioned as in disseminated sclerosis. There is a point of great interest to be worked out in regard to the possibility of hysteria—a disease of which the pathology is unknown—merging gradually into disseminated sclerosis. We are quite in the dark on this point, and this is not a fitting occasion on which to do more than advert to it. What is the connection between disseminated sclerosis and hysteria? It seems impossible to doubt, in view of the marked and almost constant occurrence of hysterical symptoms in the earlier stages of the disease, and the preponderance of cases affecting the female sex, that there is a connection of some kind, but in what that association consists I for one cannot pretend to say. No more interesting and important subject than this could engage the attention and study of our Society. The result, if one may venture to hazard an opinion founded on the experience of that which happens in regard to other diseases, would almost certainly be to refer to an organic origin many symptoms which we now term hysterical. The figure of Hysteria shrinks in proportion as the various forms of organic disease acquire greater solidity and sharper definition.

# OF THE VISUAL AREA OF THE CEREBRAL CORTEX, AND ITS RELATION TO EYE MOVEMENTS.\*

BY PROFESSOR HERMANN MUNK (BERLIN).

*Translated from the original by F. W. Mott, M.D.*

THE more vigorous the opposition which my statements regarding the visual areas of mammals have met with, and the longer the interval that has elapsed before it has been overcome, so much the more certainly, as I believe, have my views obtained a stable foundation. One important point only may be looked upon as an exception to this statement. Although it is generally agreed that to each visual area in the monkey (and in man) the corresponding halves of both retinæ correspond, and that in the dog the correspondence of each visual area is with about the lateral fourth of the retina of the same side, and the mesial three-fourths of the retina of the opposite side, the intimate connection which I have further made out between the retinal areas on the one hand and the cortical areas of the occipital lobe on the other hand, together with the so-called projection of the retinæ on the visual areas, still remains for the most part unconceded. The object of the present communication will be to further the acceptance of these views. I obtained the first insight into the subject in an early stage of my experiments. Having extirpated a suitable and not too small portion of the cortex of one occipital lobe, I found that visual disturbances occurred which were only to be explained by supposing that a second blind spot had been produced in the retina.<sup>1</sup>

\* This paper, describing experiments carried out in conjunction with Dr. Ogrégia (of Bucharest), was presented to the Royal Academy of Sciences, Berlin, on the 16th Jan., 1890, and is published in the Proceedings of the Academy.



Accordingly loss of perception corresponding to a certain part of the light-receiving elements of the retina always follows extirpation of a certain part of the cortex, and it cannot, as I have remarked, be explained otherwise, than by supposing that the central elements of the visual areas, in which the optic fibres end, and the perception of sight takes place, are arranged in a regular and successive manner like the sensory elements of the retina from which the optic fibres originate, in such a way that neighbouring retinal elements always correspond to neighbouring perceptive cortical elements.

These extremely delicate observations were, however, rendered very difficult because the disturbances were of a transient nature and the animal soon learnt to neglect the new blind spot. I have, therefore, in a later systematic research on dogs, removed large pieces suitably chosen from a visual area, alternating the anterior and the posterior, the mesial and the lateral halves and so on—and the disturbances of vision, now lasting long enough for observation, have enabled me to determine more accurately the connections of the retinæ with the visual areas.<sup>2</sup> According to the results of this research the retina may be assumed to be projected upon the visual areas in such a manner that the upper retinal zone corresponds to the anterior border of the visual area, the lower retinal zone to the posterior border of the visual area and the mesial retinal zone to the mesial border of the visual area. Finally, in so far as the retina of one side and the visual area of the other belong in the main to one another (in the dog) so does the lateral border of the retina correspond to the lateral border of the visual area (*vide* diagrams).

In conclusion, I have combined with similar partial extirpations of one visual area in dogs, total extirpations of the other, so that nothing but a remnant of one visual area remained intact).<sup>3</sup> These researches were most difficult to perform, but, when successful, the above determined projection was recognizable in the clearest and simplest manner. I have not made investigations to the same extent in monkeys. In transverse sections through the occipital



lobes the cortical convolutions (contrary to their great simplicity in the dog), here show a considerable degree of intricacy; and this has given rise to difficulties which could not be overcome. After numerous different partial extirpations, I have, therefore, contented myself with establishing the fact that, as might be supposed, *in principle*, the same projection exists in monkeys as in dogs.<sup>4</sup>

During ten years no repetition of my investigation has been forthcoming, and projection has been regarded by some as a great discovery, and by others almost as a fable. It must, indeed, seem of little worth to those who altogether dispute the dependence of the simplest visual perceptions upon the visual areas of the cortex; and it is perfectly natural that they should have failed to recognise such slight support, which even the larger extirpations in the area of the occipital lobes, have offered for the projection theory.

And as for pathological cases in man, whilst they have been forthcoming in numbers in support of my other statements in reference to the visual areas, they have seldom been favourable to the theory of projection, less by reason of the rarity of the cases than of the difficulty of observation.

Only one experimental result at last appeared in favour of projection. Of two monkeys from which Messrs. Sanger Brown and E. A. Schaefer had removed both occipital lobes, one was rendered permanently but the other only partially blind; in the latter a small part of the field of vision was left so that objects held above the level of the eyes were seen; and it was shown that in the latter case the extirpation on the under surface of the hemispheres had not extended as far forward as in the former case.<sup>5</sup> Meanwhile results confirmatory of projection have frequently occurred, unsought by me, in my numerous extirpations of the visual areas, in cases where the intended total extirpation of both visual areas had not succeeded on the one or on the other side. But I had not hitherto been able to discover any simpler or more easily demonstrated mode of experimentation than those that I have described.

Another road has recently again been opened up in the

direction of projection. Mr. Schaefer<sup>6</sup> has found that associated movements of the eyes are produced in monkeys by stimulation with the induction current of *all points of* cortex of the occipital lobes. Both eyes always move towards the side opposed to the lobes stimulated; and at the same time downwards if the excitation takes place in the upper (or anterior) and upwards if it takes place in the under (or posterior) zone of the occipital lobes: in both cases the movement (upward or downward) is strongest when the electrodes are placed on the mesial surface of the lobe or in its neighbourhood.

The intermediate zone, from which pure lateral movements were obtained, is externally wide, internally narrow, so that there is only a narrow portion on the mesial surface of the lobe.

The upward movement of the eyes was generally accompanied by an elevation of the eyelids, the downward movement by a depression, but these movements of the eyelids were as irregular in the experiments, as the changes of the pupils which sometimes accompanied the movements of the eyes. As far as could be observed the visual axes always remained parallel, when one hemisphere only was stimulated. Once, when the electrodes were placed on corresponding points of the mesial surfaces of both hemispheres, a feeble convergence of the visual axes occurred, but this result was not marked or constant enough to permit of much importance being attached to it.

We assume, therefore, says Mr. Schaefer, that these various movements of the eyes are the result of the accompanying appearances of subjective visual sensations, which the stimulation causes; and that the movements take the directions towards which those sensations are projected. Thus these experiments point to a certain connection between the parts of the cerebral visual area and of the retinae, a connection which may be stated as follows:—First, the whole visual area of one hemisphere is connected with the corresponding lateral half of both retinae, the upper zone of the visual area of one hemisphere is connected with the upper part, the lower zone with the lower part, the in-

intermediate zone with the middle part of the corresponding lateral half of both retinae. So far this is a perfect confirmation of my investigations. However, Mr. Schaefer goes further than this, and continues in the following manner:—"We may represent these relations diagrammatically by supposing the visual areas of both hemispheres to be united within the great longitudinal fissure and imagining each retina to be expanded and projected in its natural position upon the united area. If this is done, it is at once seen that the identical points of the two retinae correspond with one and the same spot of the cerebral cortex; that the upper half of each retina falls upon the upper half of the united area, and the lower half of each retina on the lower part of that area; that the inner half of the one retina and the outer half of the other, each fall upon the same hemisphere, and that a vertical zone including the centres of both retinae corresponds with the mesial part of the united area. This would connect the parts of the retinae which are concerned in direct or central vision with a part of the mesial surface of the occipital lobes, probably including a portion of the quadrate lobules. This scheme of connection between the retinae and occipital lobes differs in several points from the well-known scheme formulated by H. Munk (Ueber die Functionen u.s.w.: 5te Mittheilung) as the result of extirpation experiments, chiefly upon dogs, but partly upon monkeys. The most important differences relate to the extent of the visual area, and to the part of the occipital cortex which is connected with the central part of the retina (macula lutea). The visual area (Sehsphäre) of Munk is *confined to the occipital lobes* and the maculae luteae are *connected with the middle of the convexity of those lobes*. Consequently if the middle of the convexity of both lobes is removed central vision should be abolished. But contrary to Munk, I have not found this to be the case in monkeys. Indeed, it was the smallness of the effect produced by this bilateral lesion which first led me to doubt the correctness of Munk's scheme. On the other hand, in one experiment which I have performed since obtaining the above results from stimulation of the cortex, there was a marked defect of

central vision, produced by a bilateral injury of both mesial surfaces. Complete and permanent abolition of central vision did not result, but it was found, post-mortem, that the lesion did not involve as much of the mesial surface of the visual area, especially upon one side, as was intended at the operation. It will be necessary to follow out the question more closely by the extirpation method, and this I intend in the near future to attempt; but the difficulty of arriving at reliable conclusions from the localized extirpations of the visual area, is enormous; for the animals soon acquire the habit of compensating for local defects in the visual field by rapid movements of the eyes so as to baffle all attempts to determine the existence of such defects. I believe, indeed, that to arrive at detailed conclusions, we must await the results of perimetric observations in cases of cerebral lesion in the human subject, and that these experiments upon the visual area of the monkey are chiefly of value as affording indications of the kind of results to be looked for in man."

From this it would appear that Mr. Schaefer had mainly to do with results which were at variance with my conclusions concerning projection. The points of variance are, however, of very slender importance. I have nowhere stated that the maculæ luteæ of the ape are connected with the exact middle of the convexity of the occipital lobes. In my communication of 1880, quoted by Mr. Schaefer, I only associated the maculæ luteæ with *that* part of the cortex, which occupies *about* the middle of the convexity of each occipital lobe.<sup>8</sup> But in a communication of the year 1881<sup>9</sup> which has escaped Mr. Schaefer's notice, I have laid stress upon two experiments upon monkeys as "very interesting new experiments" concerning the cortex corresponding to the maculæ luteæ—two experiments which at least counterbalance those of Schaefer—in one of which, the convexity of each occipital lobe was extirpated about the middle, in the other a piece of the same size, situated further back and somewhat more internally, was removed.

In both, the monkey fixed its eyes *after* the operation in the same way as before, but in the latter case the visual disturbance was considerably greater than in the former;

hence I have concluded that the cortex corresponding to the "fovea centralis" is situated in the posterior half of the convexity of the occipital lobe on each side. The entire difference between us amounts practically to this: I have laid more stress on the removal of the posterior than of the inner surface, and Mr. Schaefer accentuates the removal of the inner surface. We both recognize that little importance is to be attached to the difference, since the limited number of our experiments has not permitted an accurate localisation of the cortex in question to be made.

If Mr. Schaefer, however, merely bases his scheme upon the inward displacement of the visual area, so that he unites the visual surfaces of both hemispheres on the great longitudinal fissure to a single one, and projects each retina in its natural position on this visual area as a whole—then his conclusion is not only unsupported by facts, but is demonstrably erroneous. For he quite overlooks the convolution of the cortex of the occipital lobe of the monkey, especially the *double-layer* of the cortex parallel to the surface, with the portion on the mesial border of the lobe covered in, so that the mesial edge of the cortex does not correspond to the mesial border of the lobe; he forgets that important and dissimilarly placed areas of both retinæ do not take part in binocular vision at all: he takes no account of the differences which man, the ape, the dog, &c., present in binocular vision; finally he disregards the results<sup>10</sup> which unilateral extirpations of halves of the visual areas of monkeys have afforded.

Taking into consideration all these points, which Mr. Schaefer has neglected, we can only maintain that *in principle* the same projection exists in monkeys and man, as in dogs, both in the vertical position (which as we have seen, Mr. Schaefer himself upholds) and in the horizontal position, and that the identical points of both retinæ do not correspond to the same points of the cerebral cortex; but the external half of each retina is associated with the outer half of the visual area of the same side, and the inner half of each retina with the inner half of the corresponding visual area of the opposite side. Moreover, I have observed

in an ape, in which the intended removal of the occipital lobes was not completely successful, a condition that I have often found in dogs. The ape was totally blind in the left eye, whilst sensibility to light still remained in a very limited part of the mesial and lower portion of the retina of the right eye.

Although I have not been able to pass over in silence Mr. Schaefer's difference of opinion, still at the present moment when the theory of projection has not been generally accepted, the differences between us are not so important as our points of agreement. Nevertheless Mr. Schaefer's inference, as it stands, cannot be considered as a new proof of my discovery of projection, because, to him, the supposition is based upon electrical irritation of the cortex producing sensory impressions, a theory which was indeed assumed by Dr. Ferrier, but up to the present time has remained without proof, and even without probability.

To this is to be added that no great weight can be attached to Mr. Schaefer's inference, because it only contains a further development of a point which is contained in his second assumption, namely, that the movements of the eye take directions towards which the visual sensations are projected. Nevertheless, if one is not to assume that the coincidence is altogether accidental, it must undoubtedly be accepted as affording a support for the projection theory, and as proving a close connection between the eye movements and projection.

I have, therefore, in order to clear the matter up, undertaken a series of experiments in conjunction with Dr. A. L. Obreggia, of Bucharest; choosing dogs for the purpose, because in these animals the cortex of the occipital lobes is only slightly convoluted, and therefore more easily got at, and the visual area is known better than in other animals. The dogs were placed under ether during the time of exposure of the brain, but the observations were made without any narcosis. Herr Obreggia will present an account of these experiments in full detail elsewhere, and I shall here restrict myself to a review of the experiments and discuss the results. For the topography of the brain I would refer

to the description of the dog's brain which I gave in my fourth communication,<sup>11</sup> January, 1878, and which has since found a wide circulation (fig. 3).

I count the convolutions of the convexity from the great longitudinal fissure, so that the fourth convolution surrounds the fissure of Sylvius.

As Mr. Schaefer's very serviceable experiments on monkeys lead us to anticipate, stimulation of the visual areas of dogs with an induction current brings on associated movements of the eyes towards the opposite side, both the eyes at the same time moving downwards if the anterior zone is stimulated, upwards if the posterior zone of the visual area is stimulated. The intermediary zone from which purely lateral movements originate, is but narrow; generally not half so wide as the spot A (fig. 4) of which about the middle part falls in this zone.

The upward movement is produced most markedly by stimulation of the second convolution, and it diminishes by approximation of the electrodes to the great longitudinal fissure: the downward movement shows, with the same change of position of the electrodes, no decrease, but often an increase.

The strength of the current which was found necessary for successful stimulation, is about the same as that required for the other well known cortical centres for the movements of the extremities; but for the upward movement it is somewhat less than for the downward movement. With the ordinary eye-movements are often combined movements of the upper eyelids and dilatation of the pupils, the further consideration of which need not here detain us. If one stimulates the cortex somewhat beyond the anterior border of the visual area in the region F', or beyond the lateral border of the visual area in the auditory centre B, then the eye-movements cease, if the strength of the current be not increased. These movements, which as we have seen are undeniably the results of locally restricted stimulations of portions of the visual area, are by no means slight, being sometimes even very considerable, especially on the posterior zone of the visual area. That the movements



have nevertheless escaped observation, and that even experimentors who have directed their special attention to the subject have stated that what are really the most anterior portions of the visual areas are the posterior limits for the production of the eye-movements, is not surprising to any one who considers the evolution of the study of stimulation of the cerebral cortex; it would be vain to consider all the possible causes of discrepancy. It may, indeed, be looked upon as a lucky circumstance that the correct knowledge of the subject was delayed. For had not the cerebral cortex been divided into an anterior motor and a posterior non-motor part, we should not have had the Fritsch-Hitzig experiments, and the later-acquired insight of the perceptive centres of the cerebral cortex would have met with still greater opposition than was actually the case, by reason of the short-sightedness which made all spots of the cortex that gave rise to movement when stimulated, into motor centres or connected with a great motor or psycho-motor area.

The question now arises how can we adapt these later experiments to our hitherto established knowledge of the visual centres? We may see our way to this, if we recollect the results which the total extirpation of both visual areas entails. The animal is perfectly blind, but his eye-movements are intact, the so-called voluntary, as well as the involuntary; excepting, of course, the movements which are entirely dependent upon vision, which *must* be absent in a blind animal. In the course of years I have often repeated this experiment, and I have recently convinced myself that in cortical-blind dogs and apes the eye-movements persist, even when the animal remains at perfect rest, and when every external influence is removed from his remaining perceptive faculties. The visual area has nothing whatever to do with those eye-movements of the animal which are independent of sight, neither do these movements result from excitation of the visual areas nor does the path of conduction from the place of their excitation to the periphery lead through the visual centres, consequently the eye-movements which the electrical irritation of the visual area—



induces, only correspond to particular eye-movements of the animal which are the results of visual perception.

The following offers itself as the most obvious inference. Outside the visual area and in the so-called motor area—my tactile sphere—are two spots on the cortex, the electrical stimulation of which causes eye-movements, one a spot situated in the anterior part of region F, the other, in the anterior part of region H, and accordingly as one or the other spot is excited, so will the eye muscles of the animal which are set in action, give rise to particular movements, just as by stimulation of neighbouring spots C. and D. on the cortex, arm or leg muscles will be brought into action. Therefore, if an animal makes a movement in consequence of having seen anything, it must be concluded that the excitation conducted through the optic nerve fibres to the visual areas, is transferred by associated fibres which connect these same areas with the tactile areas, at one time by this set of associated fibres, at another time by that—according to the kind of movement produced, and so through certain association fibres the excitation reaches the spots C. or D. if arm or leg movements occur: through other associated fibres the spots F. or H. if eye-movements take place. In analogous fashion the eye-movements are obtained in our case by excitation of the visual area with induction currents, since the excitation produced by the electrical current spreads to the centres F. and H. by associated fibres which run from the visual areas to those centres; whether excitation is originated by the current in the central elements of the visual area, or in the fibres themselves where they terminate in the visual area.

But against this conception the objections will naturally occur, that eye-movements alone (with at the most associated movements of the eyelids and the iris) follow the electrical stimulation, never movements of any other category, *e.g.*, arm or leg movements. No doubt an explanation of this might be found in the following consideration. It is held on good grounds, that although the excitation of a portion of the central nervous system comes into relation with different paths, nevertheless, the resistance which is

offered to its spread along the different tracts is different; and, other things being equal, the more worn certain paths of conduction are, *i.e.*, the more frequently they have already been traversed by the excitation, the less the resistance they would offer. Among all the tracts concerned in associated actions between the visual perceptive centres and the tactile (psycho-motor) centres the least resistance would probably be found along the paths for the eye-movements, since these movements are associated with vision, more often than any other. For this reason the above proposition concerning the eye-movements observed in our researches might be accepted because in these experiments the currents which were employed were very weak, in fact, scarcely more than necessary to induce movement, and the excitation was confined to as limited an area as possible.

But, if this explanation were correct, stronger stimulation should lead to movements in the other parts, the paths to which offer greater resistance, and this is never the case. After the eye-movements have come under observation, even when the electrodes are placed on the anterior portions of the visual areas and still more obviously when they are placed on the middle or posterior portions of these areas, the induction current can be very considerably strengthened before epilepsy is induced by the too powerful and ever extending excitation, and up to that point eye-movements alone occur. It must therefore be acknowledged that unless an entirely arbitrary position is taken up the eye-movements occupy an entirely special situation which could not agree with our hypothesis. A further test disproves the hypothesis by a different method.<sup>12</sup> If a frontal section be made in the anterior zone of the visual area through the hemisphere, such as I have lately recommended for the extirpation of the visual area,<sup>13</sup> only deeper, so that it opens the ventricle, and if its lower end is carried horizontally from the edge of the corpus callosum to that point in which, according to my representations, the anterior and the lateral margins of the visual area join—then, as long as the electrodes are not placed in the neighbourhood of the cut surface—electrical stimulation of the visual area produces the same movements

of the eyes after the section as before. And it makes no difference if a second section be made dividing white substance and cortex extending in a vertical continuation from the lateral or outer end of the first one, outwards in front of the descending horn of the lateral ventricle, along the convexity of the hemisphere, as far as the tip of the temporal lobe, but sparing a layer 1-2 mm. thick, lying outside the optic thalamus. This layer contains the sagittal fibres of the occipital lobe.<sup>14</sup> According to all that we know about the position of the associated fibres, the paths for associated actions have been interrupted partially by the first section, and completely by the combined sections. These tracts connect the visual centres with the cortical centres in F. or H. hence these cannot take part in movements of the eyes when these movements arise by electrical stimulation of the visual area.

No significance can be attached to the commissural fibres which pass out of the visual areas into the corpus callosum, because no idea of the process could be thus conceived, which would not at once prove untenable. Besides it is only necessary to cut through the posterior part of the corpus callosum lengthwise, either in the uninjured brain or after the before-mentioned sections have been performed, to observe that excitation will still produce movements of the eyes. Consequently, the conclusion remains that these eye-movements come to pass because the stimulation brought about by the electrical current extends to the radiating fibres of the corona radiata which go to the sub-cortical parts of the brain, and that the excitation originates in the central elements of the visual area, or in the radiating fibres where they proceed from the visual areas. The following experiment shows that this is the case.<sup>15</sup>

If a horizontal section such as I have recommended for the extirpation of the visual area be made through the hemispheres at the lateral margin of the visual area, the eye-movements will then no longer appear on stimulation of the visual area. The experiment can be made at any time, but it is best performed on the still uninjured hemisphere. The loss of blood and disturbance of the circulation of the

occipital lobes cannot be made responsible for the result since the loss is, as a rule, much less than with the frontal section before described, or when several successive sections have been made. A very interesting variation of the experiment may be performed, as follows: If, on the uninjured brain one directs the scalpel, not obliquely *upwards*, as was desirable for the extirpation of the visual area,<sup>16</sup> but obliquely *downwards*, so that its point tends towards the under surface of the corpus callosum: then the associated fibres, such as the callosal, are preserved, while the radiating fibres are cut through, and upon excitation the eye-movements are found to have disappeared. A hæmorrhage into the ventricle is not of much importance in this experiment, for a similar hæmorrhage with the frontal section, or with its extension lengthwise, does not affect the result.

To our previous knowledge of the visual areas, as central organs for vision, is therefore now added an insight into certain connections which the visual area forms with other central organs, viz., those connections which adjust the movements resulting from vision. It may be assumed, in connection with the accomplishment of these movements, that the excitation is transferred from the central elements of the visual area through associated fibres, to other cortical areas and thence to lower (sub-cortical) portions of the brain. We now know that the corona radiata of the visual area, contains, besides the optic nerve fibres, the excitation of which transferred centrally towards the visual area produces vision, also radiating fibres, the excitation of which, transferred towards the periphery from the visual area to lower (sub-cortical) parts of the brain, occasions movements, but that these are simply eye-movements the result of vision (with accompanying eyelid and such like movements), and that all other movements which are the result of vision, require the interposition of associated fibres and other cortical areas. Our experiments do not exclude the possibility that eye-movements are brought about by the latter mode as a result of visual excitation, but it is plain that certain eye-movements are preferred before all other movements which are the result of vision, in that

they are brought about in the nearest and shortest way through radiating fibres of the visual area.

There can be no doubt of the kind of eye-movement so preferred. My last communication gave me occasion to call attention to the way in which retinal and visual perceptive reflexes are to be distinguished in animals. The contraction of the pupil when light enters the eye is a retinal or optic reflex—a common reflex action for which light perception is not required, and which is brought about by means of lower portions of the brain, without the cerebral hemispheres. On the contrary there are, as I have remarked, other visual reflexes, sensory reflexes, which are effected by the action of the visual centres, *e.g.*, when—without the co-operation of the attention or judgment—the eye blinks on approach of the hand, or the animal avoids an obstacle by moving. For these visual perceptive reflexes the excitation must, we are now able to add, take a course from the visual areas through the associated fibres to other cortical areas, and through their radiating fibres to the lower centres. But the examples given, as well as the retreat from the slash of a whip, the covering at a stone being thrown, the parrying of a blow with the out-stretched arm, and the like, are visual reflexes of a higher order; reflexes which with all their manifold variety, have this in common—that they are not innate, but are evolved; and that for the commencing of the movements—involving co-operation of attention and deliberation—visual and other representations must arise. Besides these there is, we find, yet a third kind of reflex in animals which stand as it were midway between the two first kinds, *viz.*, visual reflexes of the lowest order, which are innate, and presumably never have psychological visual representations, but only have to do with the perception of light or other simple visual effects: such are the involuntary movements, which cause the eyes to wander, before being fixed upon an object which was previously indistinctly seen. It must be these reflexes (exclusively eye-movements that are the immediate and direct result of vision) for the performance of which the excitation, conducted by radiating fibres to the visual areas, proceeds again directly by radiating fibres to the lower (sub-cortical) centres.

It may be observed that we have thus obtained an insight into the course of the nerve tracts which pass out from the visual areas, without having fixed our attention upon the consequences of electrical excitation of the visual areas other than the mere appearance of eye-movements, and without having found it necessary to decide whether the excitation has arisen in consequence of electrical stimulation in the central elements of the visual areas or in the associated fibres or in the radiating fibres which pass out of the visual areas. But it stands to reason that the eye-movements resulting from stimulation of the visual areas, correspond as before stated, not simply to those eye-movements of the animal which are the result of vision, but more especially to those particular eye-movements which produce "wandering vision" and fixation of the eyes upon an object previously indistinctly seen. From the direction of the eye-movements observed on excitation of the visual area, it is therefore to be concluded that to each visual area belong the portions of both retinæ of the corresponding side which are on the same side of the maculæ luteæ, and that to the anterior, middle, and posterior zones of the visual areas respectively belong the upper, middle, and lower quadrants of those portions of the retina. Thus the theory of projection to which by the help of his assumption Mr. Schaefer has arrived in the monkey, fits in with the results obtained by our method upon the dog.

Nevertheless that result is only free from objection, if one recognises that the visual nerve fibres after their entry into the visual area, are connected directly and immediately with the central elements which serve for the perception of light; and indeed this is the simplest and most natural of all considerations which I have hitherto brought forward relating to the visual areas. This point, however, might be disputed, for it might be assumed that the visual nerve fibres enter upon their nearest connection in the visual area with the central elements of the ordinary kind, such as the lower portions of the brain and spinal cord contain; and that on the one hand connecting fibres extend out of these common reflex centres into the central elements serv-

ing for the perception of light, while on the other hand the radiating fibres of the visual areas arise out of them and conduct the stimulation towards the periphery. The results of electrical stimulation would then by no means correspond to the eye-movements of the animal resulting from vision, and accordingly would not serve as a proof of projection, but they would merely indicate (no matter whether they rested upon excitation of the common reflex centres or of the radiating fibres) that both the visual centres and their respective zones stand, for purposes as yet unknown, in different relations to the different eye-movements. To meet this objection we must again turn our attention to stimulations of the visual centres.

Roughly regarded, the eye-movements which I have above described, are the usual and regular results of stimulation; and if here and there an anomalous result occurs, yet in a general survey of the visual areas, one is satisfied to ascribe it to a spontaneous movement of the animal or to some other uncontrollable circumstance which one must always assume to occur in this kind of investigation. On a closer inspection, however, the apparently irregular exceptions are found more and more to concur; and finally it comes out as a constant and regular occurrence that under certain conditions which depend upon the place of excitation and on the condition of the eyes at the time of excitation, no associated eye-movements take place. If one stimulates the middle of the spot A (figs. 1 and 2), say of the left visual area, both eyes remain quite immovable if the dog is looking straight forward, or the left eye goes somewhat to the right whilst the right eye turns a little to the right or left; while if the dog is *not* looking straight forward the left eye always moves strongly to the right, whilst the right eye continues in this case also at rest, or only turns a *little* to the right or left. Now, as the upper eyelid is raised in all cases, and the pupil undergoes a sudden extensive dilatation, the desired certainty is obtainable that in the case of rest, or only slight movement of the eyes, the excitation was as effectual as before. If the excitation be brought nearer to the antero-lateral end of the visual area



by placing the electrodes upon the second convolution external to the fissure which divides it, or both there, and upon the third convolution—then both eyes move to the right, and at the same time more or less downwards, if the dog is looking fixedly forwards; but they converge considerably if it does not.

We have obtained more experimental results of this kind, but their conditions cannot so far be formulated with the same certainty. For the enquiry is rendered difficult by the fact that many dogs, after they are awakened from the ether narcosis, to which they are subjected for the operation, remain in constant excitement, and look continually first upon this, then upon that object, which may happen to be before their eyes. The only dogs which are perfectly serviceable for this experiment are those which are naturally patient or become quiet after the experiment has lasted a short time, so that they can be tested in a condition alternately of increased or diminished attentiveness, according to the degree of attention which is being devoted to them. In such dogs it is easy to make the observations, and they suffice to get rid of the objection now under consideration. This could not be done before, because with regard to the associated movements of the eyes, right, left, upwards or downwards, which Mr. Schaefer found in the ape, and which we have above shown to occur in the dog, there is nothing to distinguish them from common reflex movements. But the new investigations combined with the old ones, have led the results of stimulation beyond the limits of such movements. That the eyes by one and the same stimulation, at one time remain at rest, and at the other move; at one time make associated, at another converging movements, is not conceivable, if it is dependent upon excitation of nerve fibres or ordinary nerve cells. It is only comprehensible and agrees with what is to be expected if sensory perceptions are answerable for the result, if light perceptions cause the fixation of the eyes.

Other experimental results are confirmatory of these. If the electrodes, a weak excitation only being employed,



are shifted from the middle of spot A forwards and backwards (best when keeping to the second convolution on the mesial side of the fissure which divides it), the eyes move downwards the further forward one excites the area, and upwards the further backwards, and without exception the strongest downward movement that can be obtained is less than the strongest upward movement. In order to explain all this, without assuming visual perception, one must take refuge in a number of gratuitous and improbable suppositions. On the contrary, it can be easily and perfectly understood on the supposition of eye-movements which lead to the fixation of an object previously indistinctly seen, since the spot of most distinct vision in the dog is situated in the upper outer quadrant of the retina, where the tapetum is most developed. One can be in no doubt about the determination of this; and that it is right, is confirmed by further experiments. It is found, for example, in many dogs, that the strongest downward movement is not only less, but considerably less, than the strongest upward movement; and as the post-mortem section proves, there are always dogs whose tapetum, of normal size, is placed unusually high. In such dogs, the lower border of the tapetum runs at a considerable distance from the horizontal meridian of the optic papilla, while, in others, it reaches the horizontal meridian; occasionally even extending below it. Since obtaining this insight into the conditions of the retina and occipital lobe, we have been able to predicate regularly the position of the tapetum by the results of stimulation, the degree of difference between the strongest downward and the strongest upward movement being inseparably connected with the higher or lower position of the spot of distinct vision.

In the way thus marked out, by means, that is to say, of the results of electrical stimulation of the visual area, evidence of projection is obtained, which is entirely independent of the former evidence which I have given. From a scientific point of view the higher importance is undoubtedly to be attached to the earlier evidence, because the method which was employed, viz., that of extirpation,

leads directly to the point desired ; whereas by the method of excitation this is only to be attained by circuitous routes, and not without the employment of intermediate links, if indeed these are altogether reliable. On the other hand, the new evidence offers this advantage that the easier and less involved experiments, in which stimulation combined with section is employed, would, judging from the preference hitherto evinced by investigators, be more likely to be repeated than extirpation experiments, which require months at least, and are difficult alike in execution and observation. At any rate, it is of unmistakeable value, that the conformity of the experiments by both methods warrants the assumption that there is some certainty in the knowledge acquired. And this conformity extends even farther than I have yet mentioned. For the result, which the stimulation of the antero-lateral extremity of the visual area offers, is only completely comprehensible, on the assumption that the antero-lateral part of the retina of the corresponding side is in connection with that extremity, and the results of excitation of the middle of area A are only rightly to be understood on the assumption that the centre of distinct vision of the opposite retina belongs to this area ; and that the part indicated has been accurately hit upon in this latter stimulation, when both eyes remain at rest, or only one eye moves ; but not quite accurately, if both eyes move. Mr. Schaefer's endeavour to rectify projection as discovered by me, so far as concerns the horizontal relations of visual area and retina, finds therefore its own condemnation by the facts which are elucidated by this method also.

As was natural to suppose would be the case, the recent exploration in a virgin district has, besides, led to a new acquisition. That the movements resulting from vision are brought about by impulses passing along two different paths, and that the lowest visual reflexes traverse the shortest path through the radiating fibres of the visual area, deepens our insight into the structure and functions, not only of the visual areas, but of the cerebral cortex in

THE VISUAL AREA OF THE CEREBRAL CORTEX,  
AND ITS RELATION TO EYE MOVEMENTS, BY  
PROFESSOR HERMANN MUNK (BERLIN).

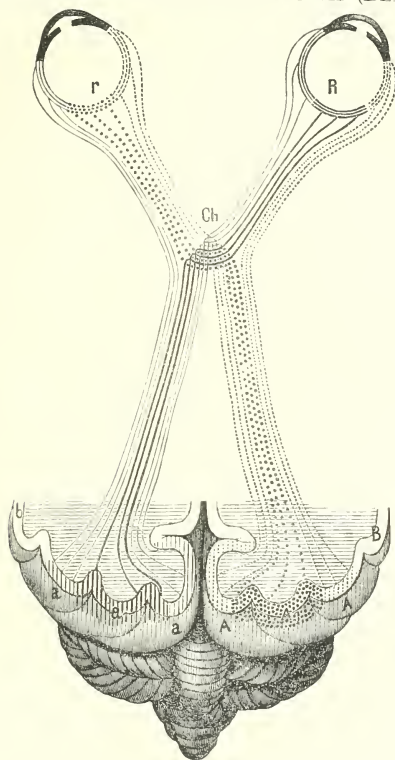


FIG. 1.

FIGS. 1 and 2 represent the connections of the visual areas with the retina in the dog (reproduced from Munk's "Über die Functionen der Grosshirninde 5te Mittheilung.")

FIG. 1 represents a frontal section made through the middle of both visual areas. The eyes and optic tracts are represented cut through horizontally.

FIG. 2 represents both visual areas as seen from above. The right visual area A is dotted. The left (a) is marked with lines, and the spots ( $A_1$  and  $a_1$ ) the extirpation of which resulted in psychical blindness, are distinguished by darker shading. The posterior surfaces of both retine are shown, and the centre of each is marked C.

B and b indicate the auditory areas at their junction with the visual area.  
Ch.—Chiasma.



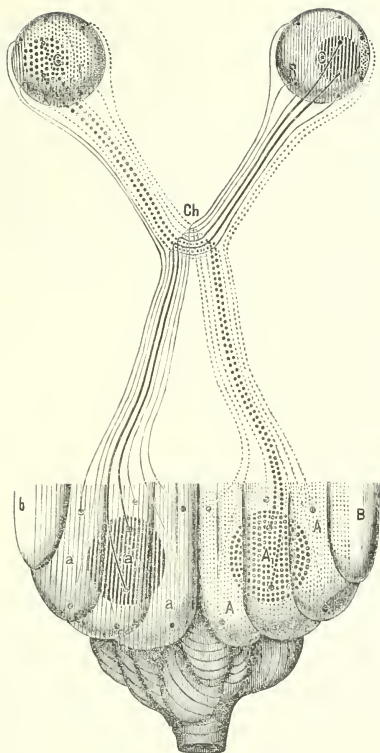


FIG. 2.

In both figures the portions of the cortex of the visual areas are connected by lines or dots with their respective corresponding portions of the retinae.

FIG. 1 shows how each visual area is connected with the lateral fourth of the retina of the same side, and the mesial three-fourth of the opposite side, the most internal fibres of the retinae passing to the most internal portion of the visual areas. The fibres coming from the central points of distinct vision are shaded darker and terminate in spots A and a.

FIG. 2.—The fibres may be traced from the upper borders of the retinae to the anterior borders of the visual areas, from the lower borders of the retinae to the posterior borders of the visual areas, and as in Fig. 1, the more deeply shaded fibres are seen passing from the centres of distinct vision to spots A and a.

With the exception of the outer portion, which is represented by lines in the left retina and by dots in the right, all the fibres proceeding from the retinae cross over at the chiasma to the visual area of the opposite side.



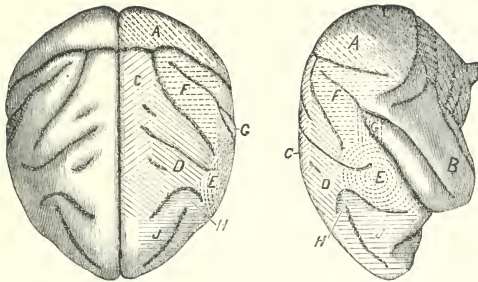


FIG. 3.

FIG. 3.—Cerebral cortex of the monkey (after Munk's "Functionen, &c., 4te Mittheilung.")

A.—Visual area.

C to J.—Tactile sensory areas—(Fühlsphaere) of Munk's, psychomotor areas of other authors.

D.—Fore limb region.

C.—Hind limb region.

E.—Head region.

F.—Eye region.

G.—Ear region.

H.—Neck region.

J.—Trunk region.

B is placed upon that portion of the cortex which from experiments on dogs is assumed to be the auditory area.

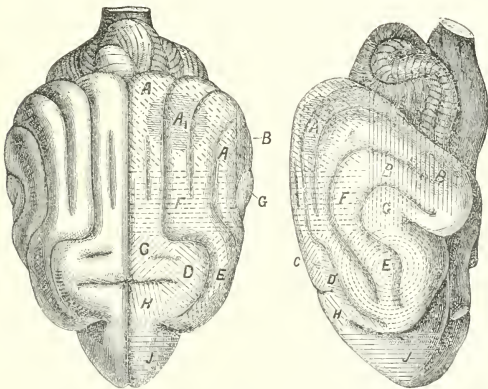


FIG. 4.

FIG. 4.—Cerebral cortex of the dog after Munk ("4te Mittheilung.")

A.—Visual area.

B.—Auditory area.

C to J.—Tactile sensory areas (Fühlsphaere).

D.—Fore-limb region.

C.—Hind-limb region.

E.—Head region.

F.—Eye region.

G.—Ear region.

H.—Neck region.

J.—Trunk region.





general, as will be subsequently shown. The projection of the retina upon the visual areas presents itself now in its full significance as the substratum for the localisation of the visual perceptions, since the involuntary eye-movements, which are brought about through the radiating fibres supply the necessary complement. Successive and opposite positions of the objects in Helmholtz's visual fields are yielded by projections, the judgment being assisted by the sensations which bring about these involuntary eye-movements, upwards, downwards, right, or left: thus projection and eye-movements together, permit such rapid and certain cognizance of the visual field, as we observe in animals, and which would be quite impossible, if it were necessary to deliberate regarding every detail in the visual field.

The discovery of the new radial fibres of the visual area will prevent the anatomical investigator from being able, without further consideration, to refer all descending degenerations which result from removal of that area to the tracts which conduct visual impressions, but there is on the other hand now opened up to him the prospect of being able to distinguish the two kinds of central elements, and of being able to demonstrate their morphological differences, a prospect which is attractive by reason of the proved connection of the radiating fibres with the light-perceiving central elements, and of the associated fibres with the representative elements (*Vorstellungselementen*).

Moreover, it is worthy of remark that our result, that the light-perceiving central elements are thrown into excitation by the electrical stimulation of the visual area, is not isolated. That the movements which result from electrical excitation of the psycho-motor area (*Fuehlsphaere*) proceed from the excitation of the grey cortex or cortical elements, has already been settled after much labour and research:<sup>17</sup> and is proved by the fact that if the electrodes lie on the uninjured surface it requires a weaker current to obtain a result, than if by the removal of the cortex the exposed white substance is stimulated; further, because the movements in the former case commence later, and last longer

than in the latter case, and lastly because, in dogs which are narcotised strongly with choral or morphia the movements disappear on excitation of the surface, not on excitation of the white substance. Corresponding statements apply also to the visual area. Here also it is found that after removal of the cortex, stronger currents are required to obtain the associated eye-movements,<sup>18</sup> than if the electrodes are placed on the uninjured surface, and it may also be shown that in dogs which are strongly narcotised by morphia or weakened by loss of blood, the excitation of the surface is unsuccessful unless a very strong current be used, whilst the excitation of the exposed white substance produces results with a current of ordinary strength. Hence it follows that the movements resulting from electrical stimulation of the visual area are derived from the excitation of the area itself, *i.e.*, of its cortical elements. One would, it must be freely admitted, need strong evidence to prove that only certain central elements of the cortex are thrown into excitation by the stimulation; evidence stronger than is as yet afforded even by all these considerations. One might, since the nerve fibres of the corona radiata are prolonged into the grey cortex, endeavour to explain the different results got from stimulation of the cortex and of the white substance, by considering that these nerve fibres have a different structure and irritability in the cortex and white substance respectively. Such a supposition is, however, constrained and unnatural, for an undoubted and great difference exists between the ganglion elements on the one hand, and the fibres on the other hand, and it is easily conceivable that the ganglion elements are excited by weaker currents, and are more easily injured by narcotics or insufficient blood supply than the nerve fibres. One is, therefore, justified in concluding from the foregoing considerations that the central elements of the visual area are excited by electrical stimulation of the same, and hence it follows that, as our above described research has proved in another way,<sup>19</sup> the excitation of the light-perceiving central elements through the electrical stimulation of the visual area represents the next advance in our knowledge.

In conclusion, I must emphasise the harmony which has existed between my earlier inquiries by extirpation and my later ones by stimulation. It goes further than projection, which was the object of our researches. It was astonishing every time that we came upon that spot of the visual area upon stimulation of which, if the dog were looking fixedly forwards, both eyes remained at rest, and, if the dog were not in this condition of fixation, the opposite eye remained immovable, how accurately this spot was found to correspond to the middle of the stelle A, *i.e.*, to the middle of those parts of the visual areas, by extirpation of which I produced psychical blindness, and which I afterwards proved to belong to that part of the retina which is concerned in distinct vision. But it is the same in respect to the borders of the whole visual area, when one takes into consideration how easily for such determinations the methods both of extirpation and stimulation are saddled with possibilities of inaccuracy, each in its own way. According to the impressions which I have formed from the excitation experiments, I should be inclined to believe that the anterior margin of the visual area, at its mesial extremity, was situated a little farther forward along the first convolution than my diagrams show; this would be in concurrence with the fact that in those cases in which the intended total extirpation of the visual area had not been perfectly successful, I had to seek for the remainder of the visual area with special frequency in the anterior mesial end of the area. On the other hand, the somewhat triangular corner which, according to my diagrams, is cut off at the antero-lateral border of the visual area from the third convolution, has now to be excluded from the visual area; manifestly, however, it is necessary for total extirpation of the visual areas to take away this triangular corner bit, in order that none of the cortex of the second convolution be left behind in the fissure between this and the third convolution.

## LIST OF REFERENCES.

<sup>1</sup> Ueber die Functionen der Grosshirnrinde. *Gesammelte Mittheilungen*. Berlin, 1881. S. 31-4; 38.

<sup>2</sup> Ibid, S. 79-94.

<sup>3</sup> Ibid, S. 104.

<sup>4</sup> Ibid, S. 38, 126-7. Du Bois-Reymond's *Archiv.*, 1881. S. 457-9.

<sup>5</sup> *Philos. Transact. of the R. Soc. of London*, vol. clxxix. (1888), B. p. 315-7; 321-3. BRAIN; a *Journal of Neurology*, vol. x. (1888), p. 370-1; vol. xi., p. 1.

<sup>6</sup> BRAIN, vol. xi., p. 1-6. Everything of importance is here quoted word for word.

<sup>7</sup> In order to make this intelligible, I must remark that Schaefer's "visual area" extends, not only over the occipital lobes, but also over the immediate neighbourhood forwards and outwards. This is of no importance as concerns projection, about which my present communication treats, therefore I shall not further refer to it in the text. But in the interest of the subject it is important in passing to throw some light upon this "important difference." In the *Phil. Transact.*, vol. clxxix. (1888) B., the conclusion to which Schaefer has arrived as the result of his and S. Brown's experiments appears as follows:—

"The two experiments, in which the ablation respectively of one or both occipital lobes was complete, are entirely confirmatory of the statements of Munk on this point, who asserts that complete and permanent blindness is produced by the removal of the occipital lobes alone without the implication of the angular gyri, and that removal of one occipital lobe causes complete and permanent hemianopsia;" (p. 321) and again, "these two experiments, namely, those in which the occipital lobe was totally removed, one unilaterally and two bilaterally, appear to afford a complete demonstration of the idea that in the cerebral perception of visual impressions, that lobe and that lobe only of the cerebral hemisphere of the monkey is concerned" (p. 322). The same position is also taken up in BRAIN (vol. x., p. 362-72). "When temporary hemiopia has resulted in consequence of extensive lesions of the temporal lobe and in one case of the angular gyrus, these transient symptoms may be ascribed rather to disturbances of the circulation in the occipital lobes and to the temporary loss of support which is afforded to that lobe, than to the fact that the visual area of the cortex spreads over from the occipital lobe into the adjoining parts of the brain. If this were the case in the monkey there should be always some remains of the visual perceptive faculty after removal of the occipital lobes alone, whereas both Munk and ourselves have found that there may be no trace left of such faculty. . . . It is, however, also possible that fibres connected with the cortex of adjoining parts of the brain (and especially of the angular gyrus), may curve backwards into the occipital lobes, and thus become cut off along with that lobe. Various facts might be cited in support of this idea. . . but we are not at present in a position to come to a definite decision upon the subject." Now the communication from which the above text (BRAIN, vol. xi., p. 1-6), is quoted, commences in the following manner:—"The visual area of the cerebral cortex in the monkey, so far as it is determinable from the results of extirpation and of electrical excitation, comprises the whole of the occipital lobe. It perhaps includes a part or the whole of the angular gyrus, although the results of extirpation would appear to show that the part played by the latter convolution is comparatively small. Removal

of both occipital lobes produce total and permanent blindness, whereas destruction of the cortex of both angular gyri is not followed by any appreciable permanent defect of vision. But, as will presently appear, electrical excitation of the posterior limb of the angular gyrus produces movements of the eyes similar to those which are produced by excitation of the adjoining portion of the occipital lobe; it is probably correct, therefore, to include at least this portion of the angular gyrus in the visual area." To this a foot-note is added. "A similar result is obtained on stimulation of the upper end of the superior temporal gyrus and of the adjacent portion of the middle temporal. This may be due, as is usually supposed, to the calling up of subjective auditory sensations; but the possibility of this part belonging also to the visual area with which it is anatomically continuous must not be overlooked." Again, on p. 4 it is stated—"I have not obtained any effect from stimulating *with weak faradisation* the anterior limb of the angular gyrus, even when well marked results have been obtained with a strength of current barely perceptible to the tongue, from the parts which have just been considered." (It is not clear what is meant by "the parts just considered;" perhaps the cortex of the occipital lobe.)

I have given in full the material for an estimation of the "very important difference," and I need add nothing to it. Schaefer must certainly have been aware that the electrical currents, when the electrodes are placed just in front of the borders of the occipital lobe, are not absolutely confined to the point which is in contact with the poles, and therefore the surrender of his earlier and correct position is only to be explained by the lively dispute in which he became engaged with Ferrier, as a result of his upholding my experiments (see BRAIN, vol. v., p. 7. 145). But I do not think that, because Ferrier, as is now universally acknowledged, at one time made certain useless experiments, more energy should be wasted in refuting his errors.

<sup>8</sup> Ueber die Functionen u. s. w., S. 127.

<sup>9</sup> Du Bois-Reymond's *Archiv.*, 1881, S. 458-9.

<sup>10</sup> Ueber die Functionen u. s. w., S. 126-7. Du Bois-Reymond's *Archiv.*, 1881, S. 456-7.

<sup>11</sup> Ueber die Functionen u. s. w., S. 62. Du Bois-Reymond's *Archiv.*, 1878, S. 552.

<sup>12</sup> The following facts concerning some similar experiments became known to us in the spring of 1889, only after we had carried out our research, and we must be content with noticing them without comment.

Schaefer (*International Journal of Anatomy and Physiology*, 1888, vol. v., part 4), removed first the one, then the other frontal lobe in front of the fissure of Rolando, cutting through the corpus callosum as well; electrical stimulation of the occipital lobe then caused associated eye-movements towards the side opposite to the excitation. "It is therefore clear," says Schaefer, "that these posterior excitable regions do not *necessarily* produce the movement in question by calling into activity the grey matter of the frontal cortex, and the centre through which they operate after removal of that grey matter, must be looked for in some more deeply seated portion of the brain (very probably in the grey matter of the corpora quadrigemina). At the same time there is no evidence that when the frontal cortex is intact they do not operate through that centre," that is to say, as appears from the context, through the anterior motor centre in the frontal cortex. Further, Danillo has stated in a preliminary communication (which is, in many respects, not very clear since we have been dependent

upon a translation from the Russian) that the associated movements of the eyes to the opposite side which he produced by electrical stimulation of the white substance of the occipital lobe in very young dogs and cats, still remain after the removal of the cortex of the anterior motor region, likewise when he separated the anterior part of the brain from the posterior by a transverse section  $1\frac{1}{2}$  cm. deep, and the same even when he made deep longitudinal sections parallel to the mesial border of the hemisphere along the first occipital convolution and also in the region of the gyrus angularis. Danillo therefore concludes that the centres for associated eye-movements are neither in the motor nor in the occipital region of the cerebral cortex, but are deeply situated. Bechterew disputes this conclusion (*Neurologisches Centralbl.*, 1889, No. 18, p. 518, Note.) According to him such centres exist both in the motor and in the occipital regions. Their presence in the occipital lobes can, he believes, be considered as proved by this—that after sections which divide the cortex corresponding to the position of this centre from the deeper lying parts, their stimulation no longer produces the customary movements, and for this he cites his “Physiology of the Motor Area of the Cortex” in the Russian *Archives of Psychiatry*, 1886 and 1887, a communication about which no mention is elsewhere to be found.

Lastly, P. Rosenbach (*Neurol. Centralbl.*, 1889, No. 9, p. 255) has stated that “the lateral deviation of the eyeball, which is obtained with certainty from the occipital lobes, and best from a particular point in Munk’s visual sphere, in all cases remains even after complete destruction of the motor region.”

<sup>13</sup> *Diese Berichte*, 1889, S. 616.

<sup>14</sup> Compare Wernicke, *Lehrbuch der Gehirnkrankheiten*, Bd. i., S. 87, fig. 45s.

<sup>15</sup> *Diese Berichte*, 1889, S. 616.

<sup>16</sup> *Ibid.*

<sup>17</sup> Franck et Pitres, *Travaux du laboratoire de M. Marey*, ann. 1878-9. Paris 1880, p. 427-29; *Arch. de Physiologie*, 3. sér. t. v. 1885, p. 7. Bubnoff und Heidenhain, *Pflüger's Arch.*, Bd. 26, 1881, S. 140-70. François-Franck. *Leçons sur les fonctions motrices du cerveau*. Paris 1887, p. 29, 35, 318.

<sup>18</sup> The occurrence, on electrical excitation of the freely exposed white substance of the occipital lobe, of associated eye-movements towards the side opposite to the excitation has already been demonstrated by Danillo and P. Rosenbach (see Note 12). Danillo has observed the movements in very young cats and dogs, hardly two months old, in which excitation of the uninjured occipital lobes was quite unsuccessful, and he has concluded that therefore Ferrier's assumption that the eye-movements which are the consequence of cortical excitation depend upon subjective visual sensations, is untenable. That Danillo's conclusion is however, incorrect, now needs no further explanation, since the radiating fibres of the visual area, which conduct the excitation towards the periphery, are affected by the stimulation of the white substance.

<sup>19</sup> *Ueber die Functionen*, u. s. w., S. 89-91; 107-8.

A CONTRIBUTION  
TO THE PATHOLOGICAL ANATOMY OF CHOREA  
WITH THE REPORT OF A CASE.

BY CHARLES L. DANA, M.D., NEW YORK.

*Visiting Physician to Bellevue Hospital.*

THE present paper deals chiefly with the pathological anatomy of the nervous centres in chorea. The changes elsewhere, particularly those in the heart have been often described, and there is a pretty definite agreement that the most uniform condition in patients dying of chorea or with chorea is fibrinous deposits on the valves of the heart, usually the mitral. Osler thinks that this occurs in 90 per cent. of cases.

I shall not take up this particular point, but turning to the nervous side of the question, I will give first the report of my own case.

Wm. G., Swiss, single, aged eighteen. Family history unknown. Admitted December 15th, 1888. Patient was always weak and sickly as a child. Has no clear remembrance of having any of the ordinary children's diseases; gives no rheumatic, venereal or alcoholic history.

Patient began to have choreic movements twelve years ago, *i.e.*, at the age of six years; cause unknown. The movements were not severe and when at the age of twelve he came to this country he was able to work and be useful.

The patient has had so-called epileptic attacks at occasional intervals for many years. The attacks used to occur at night only, but of late they have come on during the day also.

*Physical Examination.*—The patient lies in bed and is unable to walk or help himself on account of the violence of the choreic movements. He is somewhat emaciated and anæmic. His face is dull and the expression stupid, but he answers questions intelligently. The speech is slow and jerky on account of the move-



ments of the lips and tongue. Over the left parietal bone is a hole the size of a half-dollar made by a trephine in an operation performed in San Francisco fourteen months ago. No report of any hemiplegia or other form of paralysis. There are no heart murmurs, lungs normal, as are also the abdominal viscera. Urine yellow, clear, neutral, sp. gr. 1021, no albumin; microscopical examination negative. Patient's movements are so great that he cannot feed or help himself. He has several epileptoid attacks daily and nightly with tonic, and clonic and also with co-ordinate movements, throwing himself about, screaming, and frothing at the mouth. The fits are of a hysteroid character. Ordered chloral hydrate and frequent feeding with stimulants.

*December 20th*, five days later, hysteroid attacks are less numerous and only at night; choreic movements less, so that patient can feed himself. He is stupid from the chloral. Temp.  $100\frac{1}{2}$ , pulse 80, res. 20.

*December 28th*.—Thirteen days after admission, is much brighter, sits up, talks and reads quite well; chorea better. Ordered ext. physostigma gr.  $\frac{1}{4}$ . Diminished chloral. Patient improved considerably, the fits ceasing altogether, until January 6th, 1889, when he developed pneumonia and died January 8th.

*Autopsy*.—Rigor mortis well-marked, body considerably emaciated, left lung shows beginning pneumonia, heart normal; no vegetations on valves, liver and spleen normal, kidneys were small, capsule adherent and thickened; markings fairly plain, skull was extremely thick, measuring 14 ctm. ( $\frac{7}{8}$  in.) on section through occipital bone. Brachycephalic in shape. Antero-post diameter 18, transverse diameter 16 ctm., giving a cephalic index of 88<sup>1</sup>.

The old trephining hole lay over the left supra-marginal gyrus. The dura mater was not very adherent, the pia mater was thickened and congested, but not markedly adherent to the cortex. There was no excess of cerebro-spinal fluid. The brain looked normal, except for a very superficial softening seen on the under surface of both temporal lobes. The cord was apparently normal; the brain, cord, pieces of sciatic, musculo-spiral nerve and biceps were saved for further examination.

#### MICROSCOPICAL EXAMINATION.

The brain, cord, and nerves were placed in Müller's fluid, frequently changed, and at the end of three months (for cord,

<sup>1</sup> I am indebted to my house-physician, Dr. Alex. Lambert, for the above carefully taken notes of this case.



bulb and nerves), and five months (for brain) placed in alcohol. Pieces of the motor convolutions, temporal, and parietal convolutions, as well as sections of the corpus striatum and optic thalamus were cut and stained. Sections through the pons, bulb and cord were also made. The stains used were Weigert's, the ordinary hæmatoxylin, carmine, and Congo red and Golgi's nitrate of silver. By the latter method very beautiful sections were obtained.

*Convolutions and subcortical White Matter.*—There is a slight amount of lepto-meningitis, the gray matter is considerably injected, the neuroglia cells are not excessively increased. The pyramidal cells are for the most part normal. Occasionally one finds a cell undergoing pigmentary degeneration. The outlines and processes and lymph paths come out beautifully by Golgi's stain. The nerve fibres can be seen running up into the cortex; the transverse fibres are also present. The chief changes are just beneath the cortex. Here the white matter is in some sections honeycombed with little spaces, round, oval, or like long slits. The spaces are either empty or partly filled by blood vessels (which latter are shrunken by the bichromate). The blood-vessel walls are somewhat thickened, but not strikingly so. There is no real arteritis. They have, however, been tensely distended, and exudates, including red blood cells poured out. This has distended and eroded the perivascular spaces, forming large dilatations, similar to those described by Dickenson. (See illustration.)

The process, though chronic, cannot be looked upon as inflammatory, and it can only be explained by assuming a flabbiness and loss of tonus of the vessel walls (due to prolonged distension) and allowing abnormal dilatation and filtration of the vessels' contents. There is no hyperplasia of connective tissue shown by Weigert or logwood stains, nor any amyloid bodies. At the base and tip of the temporal lobe, where the surface of the brain was softened a severer grade of meningitis or chronic meningeal thickness was present and the superficial layers of the cortex showed evidences of disintegration; the ganglion cells could be seen, but the white matter beneath was more extensively honeycombed and slit, while there was evidently increase of neuroglia cells.

*Basal Ganglia and Internal Capsule.*—These parts did not appear to the naked eye so much involved as did the subcortical tissue. However, the middle portions did not harden well, and only the optic thalamus, the lower part of the internal capsule and the anterior part of the caudate nucleus could be examined

microscopically. These parts showed the same honeycombed appearance from vascular dilatation, though less marked than in sections higher up. Here also it could be seen that the small arterioles formed varicose dilatations, which were full of blood. The internal capsule showed microscopically the more serious disorganisation. Its fibres were split up by interlacing and dilated vessels, whose walls were thin, degenerated and often seemed actually falling to pieces. Here I noticed, first, a varicosity of the nerve-fibres, caused by bulging of the myeline sheath, as described by Berkley. Cross sections of the capsule stained badly with Weigert, and seemed to be full of fibres swollen or partly disorganised. There was also a decided hyperplasia of connective tissue in the capsule, which was not noted in the thalamus or caudate nucleus.<sup>1</sup>

*The corpora quadrigemina* were dotted and streaked with dilated capillaries, but showed no large perivascular spaces.

*Crura Cerebri* and III. *Nerve Level*.—There is a less degree of vascularity than at higher levels; cells of III. N. nucleus are normal, so also are the fibres of the crus and the cells of the substantia nigra.

Descending root of V. N. normal.

*Pons Varolii*.—The nucleus of the IV. N. contains few cells and some of these seem degenerated. The fourth nerves appear to have more degenerated fibres. The cells of the motor nucleus of the V. seemed to be normal in size and number, without pigmentation or vacuolation. The blood vessels are numerous and dilated, and their walls are thicker than those seen in the brain.

*Upper Medulla*.—A neuro-fibroma, the size of a pea, is seen just at the exit of the acoustic nerve on the left side, the fibres of the acoustic pass around it, a few passing into it. The only nerve nuclei in which I could detect signs of degeneration were those of the vagus (including the anterior nucleus) and accessories. The gray matter of the floor of the fourth ventricle was much honeycombed with very small cavities made by dilated arteries. There was apparently some increase of connective tissue in the pyramidal tracts. The neuro-fibroma referred to had not destroyed any parts by pressure.

*Spinal Cord*.—The pia mater was thickened, somewhat adherent and filled with distended vessels. The spinal cord showed some congestion, the distended and thickened vessels being most

<sup>1</sup> I could find no hyaline bodies such as are described by Dickenson and others.

numerous in the lateral columns. There was none of the honeycombed appearance as found in the brain and pons. In the lumbar cord a double canal was present, and for a short distance a third canal; a slight increase of interstitial tissue was observed in the lateral columns. The anterior and posterior roots showed no degenerated fibres.

*The Nerves.*—Sections of the anterior tibial nerve showed no inflammatory or degenerative changes whatever.

*SUMMARY.*—Brain, chronic leptomeningitis, non-adhesive, and therefore not severe. Diffuse and varicose dilatations of the small arteries, especially of the deeper sub-cortical matter and capsule. Degenerative changes in arterial walls; no arteritis. Perivascular lymph-spaces greatly dilated. Cortical cells in most regions normal. The severest changes, vascular, interstitial and degenerative were in the under surface of the temporal lobes in the internal capsule and adjacent parts of corpus striatum (especially lenticular nucleus), and optic thalamus (antero-internal part). Varicose nerve fibres were here noticed.

*Pons Varolii and Medulla.*—Same condition but much less marked. Cell degeneration in some cranial nerve nuclei. Slight connective tissue increase in pyramidal tracts.

*Spinal Cord.*—Slight lepto-meningitis, congestion of cord, especially in lateral tracts. Double central canal.

The record of the foregoing case naturally leads one to turn to previous accounts of autopsies for comparison. We here notice, first of all a curious distribution of these records.

The mortality from chorea seems to be much greater in England than anywhere else. In no other country have so many autopsies been made.<sup>1</sup> Of the English hospital cases 3 or 4 per cent. die. The Collective Investigation Committee found a mortality of over 2 per cent from chorea. Some years ago French physicians saw a good deal of fatal chorea, but few reports are made by them now. In Germany, there have been very few contributions to the pathological anatomy of chorea. In America I know of only three cases reported in detail.

The English observers have almost always examined acute cases, and their findings chiefly pertain to the vascular system; subinflammatory states, exudations, embolisms, &c.,

<sup>1</sup> Stuges collects eighty fatal cases reported by six English hospital physicians.

being noted. The Germans have seen more in the connective and nerve tissues, and note hyperplasia of the former with degenerative changes of the ganglion cells.

The French have as yet contributed few autopsical records of positive value.

I have examined and tabulated the *post-mortem* records in over eighty cases.

Raymond collected and tabulated (*Dict. Encyclopæd. de Sc. Med.*) seventy-nine cases. Séé in 1850 (*Memorie de l'Acad. de Med.*) collected eighty-four reports of autopsies. Eliminating the duplicated cases, I find that there are on record nearly 200 reports of autopsies made upon patients dying of, or with acute or chronic chorea.

The large majority of these are of no value whatever, so far as the observations on the nervous system are concerned. In some in which careful examinations were made, the chorea was accompanied with mania, as in Golgi's case, or was symptomatic as in Froriep's case, or was of the hereditary type, as in Macleod's.

From a critical analysis of the list of reports, I can find only thirty-nine cases which give a satisfactory account of the nervous system. Even here, in some instances, the examination is not perfectly complete.

The list of valuable records, so far as I can find them, is as follows :—

1. ROKITANSKY, *Sitz. ber. der. Wien. Akad. der. Wiss.*, 1837.
2. STEINER, *Prag. Vierteljahrsch.*, 1868, 25 Bd. iii.
3. MEYNERT, *Wien. Med. Presse*, 1868, pp. 194.
4. OPPOLZER, cited by Rosenthal; *Wien. Med. Presse*, 1868, p. 195.
5. TUCKWELL, "St. Barth. Hosp. Rept.," vol. v., p. 86.
6. RUSSELL, *Med. Times and Gazette*, 1865, p. 88.
7. E. L. FOX and R. S. SMITH, *Med. Times and Gazette*, 1870, p. 423.
8. AITKEN, *Glasgow Med. Journal*, 1853, vol. i., p. 92.
9. L. CLARKE.
10. NAUNYM, SCHMIDT'S *Jahrbüch*, Jan. 15, 1889.
11. ELISCHER, *Virch. Archiv*, vols. lxi., p. 485 and lxiii., p. 104.

- 12 to 18. DICKENSON, *Medico-Chirurg. Trans.*, 1876, vol. xli.
19. J. H. HUTCHINSON, *Phil. Med. Times*, 1876.
20. DONKIN and HEBB, *Med. Times and Gazette*, 1884, Nov. 1.
21. GOLGI, *Rivista Clinica*, 1874, p. 361.
22. BERKLEY, *Medical News*, Aug. 25, 1883.
23. ROSS, "Diseases of Nervous System," vol. ii.
24. BURY, quoted by Ross (*loc. cit.*)
25. KELLY, *Trans. Path. Society of London*, vol. xxiii., p. 94.
26. CL. DE BOYER, *Bull. Societ. Anat.*, 1875, t. xx., p. 551.
27. M. GUINON, *La France Medicale*, Jan. 19, 1886.
28. F. ROLAND, *La Progres Medicale*, 1886, vol. iv. p. 893.
29. HANDFORD and POWELL, "Brain," July, 1889.
30. Ditto ditto ditto.
31. DANA.
32. PATELLA, *Gazz. degli Ospital*, Sept., 1888.
- 33 to 39. JAKOWENKO, *Wiestnik psichiatrii i. nervopatol*, 1889, ii.

In excluding from my table so many records made, in many instances by careful observers, I do not mean to deny their value. These observations have shown in a general way that in death from chorea, congestions, extravasations, embolisms, and softenings, exist in the nervous centres. They have also proved the great uniformity of heart lesions. It is, however, only by a very minute and careful microscopical study of cases that any light upon the neuro-pathology of chorea can be obtained. For it does not follow because the cord is often found soft, or the walls of the central ventricles pultaceous, or that clots are found in the central arteries in fatal chorea that any such change occurs in the ordinary type of the disease. And the large number of incomplete autopsies upon chorea has simply confused our notions of it.

This confusion as to the pathological anatomy of chorea has been increased by the fact that systematic writers

No.	Reporter.	No. of Cases.	Age.	Sex.	Cause.	Duration.	Type & Complications.	Findings.
1	ROKITANSKY	...	...	...	.....	.....	.....	Increase of connective tissue in brain and cord.
2	STEINER	3	...	...	.....	.....	.....	Increase of connective tissue in spinal cord, with serous exudation and effusion of blood in spinal canal.
3	MEYNEPT	1	16	F.	.....	.....	Chorea of Sydenham.	Congestion and sclerotic changes in brain, colloid bodies. Degenerative changes in cells of spinal cord.
4	OPPOLZER	...	...	M.	.....	.....	.....	Increase of connective tissue in cord, softening in its upper dorsal and lower lumbar parts.
5	TUCKWELL	...	13	F.	Preceded by rheumatism.	3 weeks.	Chorea of Syd.	Emboli in both posterior cerebral arteries and partial softening in under surface of temporal lobes. Spinal cord normal.
6	RUSSELL	...	14	M.	Rheumatic.	About 1 month.	Chorea of Syd.	Brain atrophy, increased subarachnoid fluid. Flat ocellomyones.
7	E. L. FOX	...	17	F.	Fright. ...	1 month.	Chorea of Syd.	Meningeal hemorrhage, cord and corpus striatum examined only.
8	AITKEN	...	17	F.	.....	10 days.	Sub-acute chorea, inebcility.	Small vessels of corpus striatum engorged with blood. Other parts not examined.
9	L. CLARKE	...	...	...	.....	...	...	Varicose dilatation of blood vessels of gray matter of brain.
10	NAUNYM	...	17	F.	.....	6 years.	Severe type of chronic chorea.	Lessened spec.-grav. of basal ganglia.
11	ELISCHER	...	22	F.	3rd attack. ...	4 months.	Chorea of pregnancy.	Oedema of brain and cord. Brown-red coloration of pia in fissure of sylvius and at base, caused by fungi growing along the vessels.
12	DICKENSON	8	10	F.	Rheumatic. ...	3 days.	Chorea of Syd.	Diffuse irritative process causing increased proliferation of nuclei of neuroglia and of vessel walls. Signs of degenerative process in nerve cells.
13	Ibid	2	10	F.	Fright, 3rd attack.	3 weeks.	Chorea of Syd.	Hyperemia of parts of brain and especially of cord. Hydropyelia, especially of lower cord (congenital?) Hematomyelia of upper part of cord.
14	Ibid	3	7	F.	?	24 days.	Chorea of Syd.	Distension of arteries and veins and effusion in perivascular spaces. Congestion of cord.
15	Ibid	4	8	F.	Fright. ...	2 months.	Chorea of Syd.	Distension of arteries and veins, especially of optic thalamus, congestion and extravasation in cord.
16	Ibid	5	11	M.	Rheumatism.	6 weeks.	Chorea of Syd.	Congestion of brain and cord.
17	Ibid	6	13	F.	Rheumatic, 3rd attack.	3 weeks.	Chorea of Syd.	Congestion of corp. striatum and in the white matter of one of the convolutions with signs of small embolisms? Congestion and sclerosis of gray matter of cord.
18	Ibid	7	54	M.	?	4 years.	Chronic chorea.	Congestion of basal parts of brain and old spots of sclerosis? especially in motor convolutions. Congestion of cord.
								Congestion of brain with old spots of so-called sclerosis (arteriofacts?) especially in basal parts and corpora striata.

No.	Reporter.	No. of Cases.	Age.	Sex.	Cause.	Duration.	Type & Complications.	Findings.
19	J. H. HUTCHINSON	1	12	M.	Fall. ...	3 weeks.	Chorea of Syd.	Congestion of brain with some plugging? of arteries of corpora striata, cord is soft, 3rd to 5th dorsal.
20	DONKIN & JEBB	.....	20	F.	Fright. ...	7 days.	Chorea of Syd. (?) with mania.	Brain very soft, walls of vessels disintegrated, cord showed congenital abnormalities, blood vessels full of micro-organisms.
21	GOLGI ...	.....	42	M.	.....	10 years.	Chronic symptomatic, with mania.	Pachy and leptomeningitis, atrophy of cerebral convolutions white matter hyperaemic, spots of softening and gray degeneration, corpora striata softened, cord soft, membranes thick, ganglion cells atrophied, varicose axis cylinders, hyperplasia of connective tissue in cord and cell pigmentation.
22	BERKLEY... ..	.....	41	F.	Fright. ...	7 years.	Chronic chorea, imbecility.	Dilatation and thickening of arterioles with necrotic areas about them, degenerative changes in ganglion cells, varicosities of nerve fibres, amyloid bodies. Same changes very much less marked in bulb. Same changes quite marked together with sclerotic changes in cord, no embolisms.
23	ROSS... ..	.....	...	...	.....	.....	Chorea of Syd.	Hyperaemia of spinal cord, especially of anterior and anterolateral arteries. Degeneration of accessory nerve cells.
24	BURY ... ..	.....	...	...	.....	.....	Chorea of Syd.	Periarterial exudations and erosions; and lesions in general like those described by Dickenson.
25	KELLY ... ..	.....	9	F.	?	15 days.	Chorea of Syd.	Soft spots and small extravasations of blood in corp. striat. Fatty degeneration of capillaries and small arteries.
26	CL. DE BOYER	.....	7	F.	.....	4 months.	Chorea of Syd.	Marked meningo-encephalitis and cerebral atrophy, other parts normal. Microscopical examination (?)
27	GUINON ... ..	.....	19	F.	.....	2 months.	Chorea of Syd.	Nothing.
28	ROLAND .. ..	.....	7	F.	Scarlet fever.	Acute.	Chorea of Syd.	Very great cerebral hyperaemia, two pseudo cysts of the choroid plexus.
29	H. HANDFORD & E. POWELL	.....	10	M.	.....	21 days.	Chorea of Syd., with insanity.	Intense cerebral hyperaemia, thromboses and minute haemorrhages in pons and upper cord.
30	H. HANDFORD & E. POWELL	.....	20	F.	.....	2 weeks.	Chorea of Syd., with insanity.	Same as No. 24.
31	DANA ... ..	.....	19	M.	.....	12 years.	Chronic with epilepsy.	Vascular dilations and perivascular erosions.
32	PATELLA... ..	.....	11	...	.....	.....	Chorea of Syd.	Softened areas subcortical, especially in occipital lobes. Hyperaemia, embolisms (?)
33	JAKOWENKO ...	6	...	...	.....	.....	Chorea minor, later delirium acutum.	Small hyaline bodies in globus pallidus lying about vessels, vide Meynert, Dickenson, Berkeley.



have usually cited all the various findings in fatal cases as evidence of the lack of any definite anatomical basis of the disease. In fact the records in sub-acute cases, in chronic cases, in juvenile or adult cases, and in complicated cases, should be studied separately, and compared together with the facts of the clinical history kept in view. In this way, it will be found that there is very much less discrepancy in results than is supposed.

In the lists of fatal cases collected are :—

1. Sub-acute chorea of Sydenham.
2. Chronic       ,,               ,,
3. Hereditary   ,,       (Huntingdon's).
4. Congenital   ,,
5. Symptomatic ,,

It is only the first two classes of cases that form the chorea as ordinarily met with. It is this disease whose pathology and anatomy is here specifically sought, and whose autopsical records I have collected.

I shall analyse my table with reference—

1st. To sub-acute chorea :

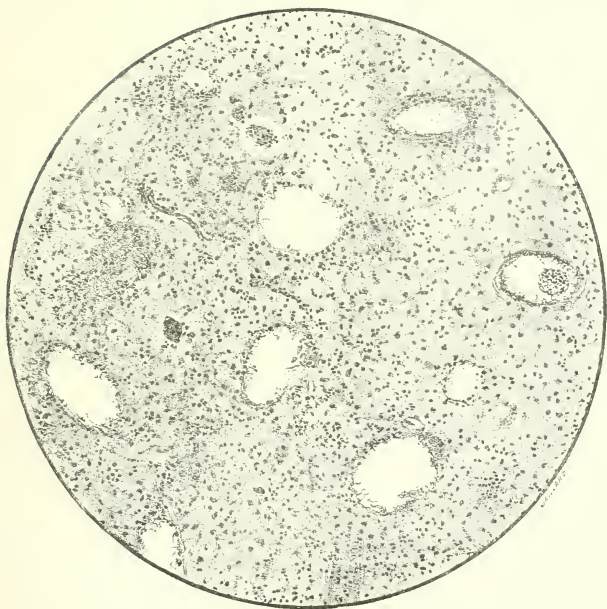
Among the thirty-nine cases there are twenty-five in which the disease was the sub-acute chorea of Sydenham, uncomplicated with insanity, or other serious disorder. Leaving out the six cases of Jakowenko, which are published in Russian, and not accessible in detail to me, there remain nineteen cases, in which careful microscopical examinations were made. In sixteen of these there is a record of intense cerebral hyperæmia, periarterial exudations, and erosions, softened spots and minute hæmorrhages, and occasionally embolisms. The changes are most marked, not in the meninges, but in the deeper parts of the motor tract, the lenticular nucleus and inner parts of the thalamus.

In a few of the cases, only, is the cord affected somewhat similarly.

Of the remaining three cases, in one (Roland's) absolutely no changes are recorded. I cannot but think that they were overlooked, or that a mistake was made in some way.

In one case, only the corpus striatum and cord were examined, and here spots of softening were found ; and in one







case, the brain was very soft, the vessel walls were disintegrated, and full of micro-organisms.

In two cases of sub-acute chorea with insanity, there was intense cerebral hyperæmia with thrombosis and minute hæmorrhages, much as in uncomplicated cases.

In the six cases of Jakowenko, he says nothing about congestion, but describes minute hyaline bodies, found chiefly in the inner divisions of the lenticular nucleus (*globus pallidus*).

## 2nd. Chronic chorea.

There are records of five cases of chronic chorea, lasting from four to twelve years.

Here we find dilatation and thickening of arterioles with necrotic and erosive changes about them, degenerative changes in ganglion cells, varicosities of nerve fibres, old spots of sclerosis, and hyaline bodies.

The cord is somewhat similarly involved, but to a less extent.

There are no evidences of inflammation, or of continuous humoral irritations, such as eventually lead to decided arterial thickening.

Summing up we find that in the subacute types of chorea there is a hyperæmia of the brain and parts of the cord. In the brain this is not meningeal but subcortical and basal. The arterial walls are paralysed, dilated and badly nourished, so that exudations occur, and the lymph spaces become distended and eroded. There is sometimes stasis, thrombosis and spots of softening, or the walls give way, and there are minute hæmorrhages. The lymph spaces around the ganglion cells are not dilated.

In older cases, the vascular and neuro-degenerative changes are marked. The small arteries are permanently dilated, a little thickened and degenerated, perivascular channels may be more eroded and distended.

There is now some connective tissue proliferation, and signs of degeneration in the ganglionic cells. The nerve-fibres show a varicosity. (Berkley's case and my own). Hyaline bodies are seen.

In fine, we have in chorea, first a vaso-motor paralysis

and trophic disturbance, affecting certain areas of the brain, and to a less extent of the cord. Then we have this becoming chronic, with connective tissue, hyperplasia and degenerative changes in ganglionic cells and fibres.

As for the localisation of the lesion, the earlier English writers all looked for it in the basal ganglia, and their findings are, perhaps, a little biassed by the expectation of seeing something in these localities. In some autopsies only the cord and basal ganglia were examined. We cannot, however, locate chorea in these places exclusively, nor in the globus pallidus, nor in the cortex. It is rather a disease of the intracranial motor tract, including its starting point in the cortex, and especially in its co-ordinating adjuncts, the lenticular nucleus and thalamus.

# NOTES ON A CASE OF TUMOUR OF THE CEREBELLUM WITH AN ABSENCE OF ALL SYMPTOMS.

BY P. WM. MACDONALD, M.D.

---

(Read at a meeting of the Dorset and West Hants Branch of the British  
Medical Association.)

In placing on record the following facts and notes my object is two-fold. Firstly, to show the value of negative facts and secondly, to prove the existence of a tumour in the left cerebellar hemisphere, without even a solitary symptom.

The respect due to old age may be said to reach the "zenith" of its depth and sincerity, when at the close of four score years and nine, a hitherto unknown mine of pathological wealth is discovered.

M. A. H. was admitted into the Dorset Asylum in December, 1864. From the Case Books and Registers, I learn, that her case was an ordinary one of maniacal excitement and mental perversion with many fixed and absurd delusions. During her residence in the asylum—which extended over a period of twenty-four years—her mental state drifted slowly towards the goal of dementia, in which condition it was for many of the latter years of her life. At the same time, though her mental state was one of dementia, she was conscious of all her surroundings and could answer questions till the last. One of her favourite themes of conversation was to make a few most affectionate enquiries after your health, and then, without any warning and before that you had time to move, she would pour forth in rapid succession, chains of the most obscene and disgusting language possible to imagine. Such are the vagaries of the human mind, which, I mean the mind, is ever an uncertain quantity, and of questionable quality when the higher or controlling bricks are from whatever cause disarranged, disorganised, or generally out of repair.

This poor woman was a member of the bronchitic family, and

often have I heard her say: "Doctor, cannot you cure the bronchitis?" Her appeals were not to be gratified, for on the 21st day of June last did this same bronchitis manifest its existence by an unexpected and grand display of renewed activity. No sooner had the fire been well ignited than the general grate began to fall to pieces, and seven days thereafter, or on the 28th day of June, did this same fine old specimen of eighty-nine human summers join the majority from natural causes, to wit, bronchitis. Into these few sentences I have squeezed a life record of four-and-twenty years.

My interest in the dear old woman did not lessen notwithstanding her disappearance from all that was mortal, and I respectfully followed her body to the mortuary. I will now tell you what we found there, or to be more precise, relate to you what careful dissections revealed.

In the thorax we found a comparatively healthy heart with a dilated aorta. The lungs were old offenders and everywhere we found the changes incident to long-continued bronchitis. Posteriorly and at the base the lung tissue was engorged and the larger and middle-sized bronchi were congested and contained thickish secretion. The abdominal organs, with the exception of the liver, were fairly healthy, but slightly congested. The liver was of an irregular shape, with rounded edges, pale, soft, and friable. On section hyper-glistening and a typical specimen of slow fatty changes. There was a small intra-mural uterine fibroid, and the gastro-intestinal tract was normal. We now proceeded to examine the head, and after removing an unusually soft and porous skull-cap, we came to a pale, thickened dura-mater with many adhesions to the cranium. The arachnoid was milky. No excess of subarachnoid fluid. Pia mater attenuated but no pia cortical adhesions. On cutting through the tentorium cerebelli on the left side we observed that the left hemisphere or lobe of the cerebellum was pressed upon by a round greyish coloured tumour, the size of a small apple, softish on the outer side, and adherent to the occipital bone. The surface of bony adhesion was the size of a shilling and the bone was simply a little rougher than natural. This was the only attachment to bone.

The brain removed it was noticed that the grey matter of the cerebrum was of an ashy-grey colour, while the central medulla was anæmic. Various ganglia normal. Arteries extensively diseased, being thickened and calcareous. The corpus callosum was softened. Taken as a whole the brain was wanting in consistence and volume. No special localised softening or lesion other than the cerebellar tumour.

Turning our attention to the tumour we note that it is a round encapsulated growth pressing on and causing atrophy of the left cerebellar lobe. In all probability it had its origin in the dura mater, and the attachment to the bone was of secondary occurrence and what we should expect to find.

In its development the main and principal increase of structure was towards the cerebellum, and it could not have had much tendency to grow in an outward direction, for its attachment to the occipital bone was only sufficient to produce an unnatural roughening on the surface of the internal table. Except at the point of origin where it is rather soft, the tumour is firm and compact, and from its general character will be placed among the sarcomatous tumours. Considering the size of the tumour, the small degree of displacement or destruction of the cerebellum is remarkable.

Microscopically the tumour is formed of small spindle nucleated and granular cells, and many numerous cellular and fibrillated elements. The somewhat rare appearance of the spindle cells being arranged in concentric nests was observed. The tumour then is a small spindle celled sarcoma.

So much for the characters and nature of the tumour, and next as to its presence and yet no symptoms. At no time during the patient's life in the asylum, nor before as far as I know was there any symptom of cerebellar disease.

We are told that extensive and even gross destruction of the lateral lobes of the cerebellum, may take place without producing any obvious symptom. The case under discussion must be placed among the unknown or undiagnosed but, though this has to be admitted, we may not unprofitably review and compare the material at our command with recorded facts and opinions.

By a process of slow and gradual pressure the tumour caused the destruction of at least one-third of the left hemisphere of the cerebellum. This depression in the cerebellum which was the result of atrophy (from pressure) proceeded in a central direction but not as far as the middle lobe, thereby leaving this the most important lobe of the cerebellum unaffected. The cases are few and rare where symptoms are absolutely wanting. In writing of this class of cases Dr. Byrom Bramwell says: "We must conclude then, that in some rare cases (in which an intracranial tumour is stationary or of extremely slow growth; in which there

is no increase of intracranial tension; and no irritation in the neighbourhood of the lesion) all symptoms may be entirely wanting." In the present case there were not even transitory symptoms to lead us to suspect an intracranial growth, and mainly for this reason the eyes were not examined. Though I can vouch for the absence of two (almost pathognomonic) symptoms, viz., headache and vomiting, I cannot definitely state that a third, viz., optic neuritis, was not present. The patient's sight was above or better than the average at 89. With the aid of cheap spectacles she could read small print. But this is no proof of the absence of optic neuritis, for it is now an accepted fact that optic neuritis may be associated with perfect central vision. Therefore it is to be regretted, that the eyes were not examined; at the same time, I am of the opinion that neither optic neuritis nor atrophy existed.

I do not think that a lesion in the lateral hemisphere of the cerebellum will *per se* cause any symptom by which it can be recognised, and the present case supports this view which is the opinion of our best authors on nervous diseases. Dr. Ferrier says: "the functions of the lobules of the lateral lobe of the cerebellum are principally concerned in various mixed movements of the eyes and head." This he has demonstrated by experiment and adds "it is only when the disease has been of a comparatively limited extent or confined to one lobe that there has been an absence of the characteristic symptoms."

Dr. Gowers says: "the hemispheres may be destroyed without recognisable symptoms, but this is not true of the middle lobe."

In the case under consideration we had actual loss of substance in the left lobe, but no interference with the functions of the middle lobe, hence no recognisable symptom. We can understand the presence of a small lesion and no symptoms, but when we meet with a large tumour—(such as I have shown you to day)—causing gross destruction, and still no symptom, we are constrained, to ask the question—are the accepted views and theories regarding the relations of the grey nuclei and peduncular fibres (in the lateral lobes of the cerebellum) to be relied upon or do they require revision and confirmation?



I have stated that at least one-third of the left hemisphere had undergone atrophy from the presence of the new growth, and as a result, we have the "semi-lunar tract" obliterated, and the outer margin of the "corpus dentatum" compressed, yet withal no symptoms.

From these facts we might build the hypothesis that the hemispheres of the cerebellum are mainly subservient to psychical processes and that a large lesion may exist without producing any recognisable symptom so long as the middle lobe is not compressed.

I invariably advocate the recording of negative facts, believing as I do, that such facts are often of as much value as positive ones, in elucidating and solving all the mysteries surrounding the functions of the greater and lesser brain.

The hypothesis that the cerebellum presides over the functions of organic life was we are told, founded on false anatomical views, and "there is indeed little or no evidence that the cerebellum is essential to the due performance of the functions of organic or vegetative life." (Dr. Ferrier).

"As we descend the scale the hemispheres of the cerebellum lessen, until they disappear in birds, in which the whole cerebellum corresponds to the middle lobe of man."

"Moreover the cerebellar hemispheres have this in common with the parts of the cerebral cortex with which they are connected, that simple loss of substance causes no definite and recognisable loss of any function of the brain." (Dr. Gowers).

Finally, "The effects of injury and disease show that the functions of the cerebellum are outside the sphere of mind proper, as expressed in sensation, emotion, volition and intellect." (Dr. Ferrier).

One word in conclusion. Was the tumour in this case the cause of the mental disease? No. I do not think it was the cause, but it may have been a secondary and irritative cause, and therefore, cannot wholly be raised from the platform of cause and effect. This view is the more tenable if we place any credence in the old theory that the cerebellum is in some way connected with psychical processes.

*October, 1889.*

## CASE OF HÆMORRHAGES IN AND ABOUT THE PONS.

BY J. S. RISIEN RUSSELL, M.B., AND JAMES TAYLOR, M.A, M.B.

WE are indebted to Dr. Ramskill for permission to use the following case, which, on account of its rarity and the many points of interest connected with it, seems worthy of being placed permanently on record.

The patient dated the commencement of his illness from January, 1887, when he began to feel weak, to lose flesh, and to suffer from frequent attacks of frontal headache. He continued in the same somewhat indefinite condition for a year. At the end of that time he had a sudden loss of power in the right side, accompanied by sickness and vomiting, but without loss of consciousness. This passed off sufficiently in three days to permit of his walking about as usual, but from that time he remained weak. He had two attacks of giddiness subsequently—one two months later and the other in December last, without loss of consciousness or vomiting, but with a subsequent loss of ability to talk—in one case for four hours, and in the other for half an hour. There was no increase of weakness after either of those attacks. During the time he had been ill his memory became very defective, and from being an active, busy man, he became dull and apathetic.

When he came under observation at the National Hospital for Paralysis and Epilepsy, Queen Square, in April, 1889, he presented general weakness in all four limbs, more marked in the right arm and leg, and his gait was slow and somewhat tottering, but without any distinct hemiplegic character. There was no asymmetry of the face, and the tongue was protruded in the middle line. His utterance

was somewhat blurred. He understood what was said to him, and although he could not give a reliable history of his illness, apparently from impairment of memory, he was quite aware of his condition and surroundings. He was very drowsy, and it was difficult to fix his attention for more than a few minutes at a time.

A week after admission he was noticed to have lost almost entirely the power in his left arm and leg, while the limbs on the right side remained practically as before. Both sides of the face were affected, but the right much more than the left. The tongue was protruded to the left, and the articulation was more indistinct than formerly. He gradually after this became more and more drowsy and lethargic, and passed his evacuations in bed. He could still protrude his tongue, although he required a good deal of pressure to make him do so. He had considerable difficulty in swallowing, or rather a tendency to allow the food to remain in his mouth as if it were too much trouble to get it further. There was no change in the paralysis, but he gradually passed into a condition of coma, and died with a high temperature three weeks from the date of admission. He had no ocular paralysis and no optic neuritis, nor had he anything in the nature of a fit, except the three attacks already mentioned—one at the commencement of his illness and the others subsequently. There was slight albuminuria present, and evidence of a hypertrophied left ventricle.

It will be seen that this case presented some difficulties in diagnosis. Evidently there was some condition causing interruption of the tracts at some point between the cortex cerebri and the spinal cord, but there was difficulty in defining firstly, the site of the lesion, and secondly, its nature. An ordinary cerebral hæmorrhage first into one side of the brain and then into the other, was improbable, because in the first place there was no loss of consciousness at any time, and in the second, the recovery from the first attack was more rapid and probably more complete than is usually the case. Yet the presence of kidney disease and a hypertrophied left ventricle made it likely that the condition of arteries was that usually associated with cerebral hæmorrhage.

Tumour naturally suggested itself as a possible explanation of the symptoms, for these were ingravescent, and there was much headache and drowsiness. To cause the symptoms a tumour would require to be situated at a point where the tracts of opposite sides approach each other more or less closely. The localities in which the tracts are in such proximity as to be liable to pressure by a single growth are the medulla, the pons, the crura and the internal capsules. A tumour of the medulla such as to give rise to the condition of the extremities which was present here could hardly exist without producing symptoms of pressure on some cranial nerves, and the same might be said of the pons, where a growth of such extent as to cause the degree of paralysis here present would almost certainly give rise to oculo-motor paralysis.

Again, a tumour affecting both crura was excluded by the fact that a tumour large enough to cause the amount of paralysis present here would almost certainly have involved one, if not both, third nerves. Besides, as the face fibres in the crura are internal to those for the arm and leg, it is evident that a tumour pressing on both crura would first and most severely affect the face fibres, while the affection of the extremities would be secondary and less severe. The very opposite was the case here.

The only remaining point where the fibres of the motor tracts are in reasonable proximity is in the internal capsules, and a tumour pressing on both capsules seemed a feasible hypothesis to account for the symptoms. Dr. Bristowe in his recent volume on 'Diseases of the Nervous System,' relates five cases in which pressure was exerted at this point, in four of them by a tumour of the corpus callosum, and in the fifth by a growth starting in the lateral ventricle, and involving the corpus callosum secondarily. He gives as the characteristic symptoms of such tumours (1) headache and somewhat vague symptoms of progressive cerebral disease; (2) gradual onset of more or less well-marked hemiplegia; (3) appearance of similar symptoms on the opposite side of the body; (4) coming on of dementia with loss of speech, difficulty in swallowing, and want of control over rectum

and bladder. In this case all those symptoms were present, the only point in which a difference could be found being that, although the other symptoms were of gradual onset, both the initial right hemiplegia and also the later left one occurred suddenly.

The presence or absence of optic neuritis might be expected to help in the diagnosis of such a case, but unfortunately it is not so. Of the five cases related by Dr. Bristowe of tumour in or involving the corpus callosum, in two neuritis was present, in one it is noted as being absent at least ten days before death, while in the others it is not referred to. So that the absence of this condition in the present case (and there was no trace of it half-an-hour before death), did not give us much help. Nor could the absence of epileptiform convulsions count for anything, as in only one of Dr. Bristowe's cases were fits present.

The only alternative to such a tumour seemed to be some lesions in the pons probably of the nature of softening from occlusion of vessels. The suddenness of onset of the two attacks, the fact that after the second the right side of the face and the left side of the body were the parts most affected, and the absence of the peculiar mental change described by Dr. Bristowe in cases of tumour of the corpus callosum, were all in favour of lesion of the pons and against the probability of such a tumour. The idea of hæmorrhage into the pons at more than one point had occurred to us only to be discarded. As a rule one hæmorrhage into the pons kills almost at once, and it seemed most unlikely that two or more should take place into one pons giving rise to symptoms spread over two and-a-half years. Yet at the autopsy this was found to have taken place. There were in the pons hæmorrhages in such positions as to affect the tracts on the two sides, of which a more detailed description is given below. The corpus callosum was healthy, as also were the capsules and basal ganglia. The arterial walls were much thickened and the left ventricle was hypertrophied.

G. B., æt. fifty-three, married, policeman, admitted April 3rd, 1889; died April 24th, 1889.

Two years ago last January patient began to feel weak and to

lose flesh; continued to do so for a year. No definite symptom except occasional frontal headache. At the end of this time while at dinner one day he became sick, vomited, and would have fallen from his chair. He did not lose consciousness, and no twitching of limbs or face was observed. Immediately after, however, right arm and leg were found to be powerless, and face was drawn to right. Next day had severe headache, but had recovered so far as to be able to use knife with right hand, and two days later had so far recovered the use of his leg as to be able to walk downstairs. He has continued weak on right side since and has suffered a good deal from frontal headache.

Two months later, while walking with his wife, became "giddy" and caught hold of her. Had no loss of consciousness and did not vomit, but he was unable to speak for four hours. The right arm and leg seemed no weaker after this.

In December last had another attack similar to last. He was in bed, felt giddy; did not lose consciousness, was not sick; had no twitching, face was not drawn, did not lose any more power. Was unable to speak for half-an-hour.

His memory has been gradually failing during his illness, and during the last nine months he has become very stupid. Frequently takes no notice when spoken to, but has never been known to ramble. Has had almost constant headache, but only sickness once in addition to the occasion already referred to. This was in the out-patient room of the hospital seven or eight weeks ago.

During the last year has had frequency of micturition, and inability long to resist the call, as well as loss of control over liquid motions.

His previous health was excellent, and he was regular in his habits. He neither drank nor smoked, and his family history shows nothing noteworthy. He denies venereal disease.

On admission patient is seen to be big, strong and healthy in appearance. There is distinct, although by no means very great right-sided weakness, affecting arm and leg, but not the face. The tongue is protruded straight, and the palate is unaffected. Sensibility is normal except that touches on right upper arm and forearm are referred to a segment below, while those on back of hand and fingers are referred to a point slightly higher. Touches on left upper limb are referred to about half a segment below. Sense of position of right arm is defective, of left is accurate.

Legs are well-nourished but feeble, right being the weaker. Sensibility to touch, or sense of position are perfectly normal.

His gait is feeble and tottering, but not characteristically hemiplegic. Knee jerks are active and equal; ankle clonus is well marked on both sides, and the plantar reflex is much exaggerated and equally so on the two sides. The wrist jerks are equally well marked; the triceps is rather more marked on right side.

The pupils react normally, there is no ocular palsy, and the optic discs and fundi appear normal. Taste seems somewhat blunted, equally so on the two sides. Smell is acute.

There is evident hypertrophy of the left ventricle, and a slight systolic murmur accompanying first sound at the aortic cartilage where the second sound is sharp. The urine has a specific gravity of 1018, and contains a distinct trace of albumen.

As to his psychical condition, he is dull and slow in comprehending, his memory is bad, articulation is thick, and he has difficulty in finding words. He understands what he reads, and also what is said to him, but he takes some time to do so.

Patient remained in the same condition during the first week of his stay in hospital. On the morning of April 10th he was observed to be very weak on left side, and on examination the arm and leg on this side were found to be paralysed. He could only just move the hand, he could not raise it to his head, and his leg was completely paralysed. The tongue was protruded to the left, but the mobility of left side of face was distinctly greater than that on the right side. The temperature was normal. The speech was rather more indistinct than formerly. During this and several succeeding nights, and also sometimes during the day, it was observed that the respiration was of the Cheyne Stokes variety, but occurring during natural sleep. It began as gentle respiration, gradually increasing in force, then dying away again until there was entire cessation for about twenty to thirty seconds.

Patient gradually became more and more stupid, began to pass his evacuations in bed, and to have difficulty in swallowing. The right side also seemed to become more feeble. The optic discs remained distinct and the margins well defined.

On April 23rd the following note was made:—"Patient takes still less notice to-day. He opens his eyes when one goes to his bed side; when asked to put out his tongue takes no notice. Blinks when hand brought before either eye. Can be made to hold up right arm very slightly when it is passively raised from the bed. Does not take any notice when pricked on arms or legs, but does if pricked on his face. Plantar reflex brisk, left greater. Knee jerk not so active as formerly, left being greater; ankle clonus well marked on left side, slight on right. Temperature



this morning is 102°. Urine acid, 1026, no deposit; marked cloud of albumen, no sugar. Passes evacuations in bed. Mr. Gunn examined eyes to-day and described discs as hyperæmic, the inner margin indistinct, but no effusion or other sign of inflammation."

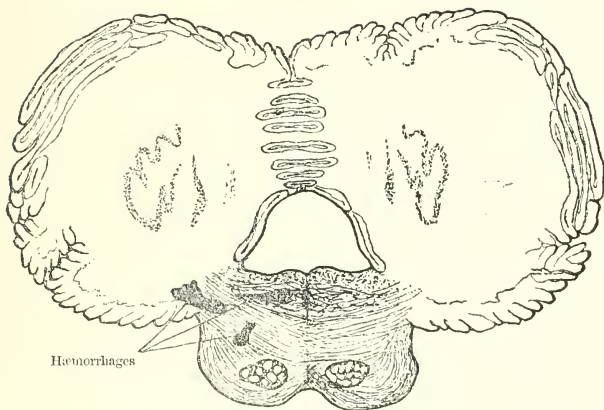
Patient gradually sank into complete coma and died on the day after the foregoing note was made. Temperature rose to 104° before death.

*Post-mortem.*—Heart was found to be large, left ventricle very much hypertrophied, valves all competent. Very slight atheroma of aorta. The vessels generally, but especially those in the brain, were very much thickened, and atheromatous patches were evident in several places. The kidneys to the naked eye appeared fatty. The capsule stripped off readily. The left had a urine-containing cyst about the size of a large marble just under the cortex. The other abdominal and thoracic organs appeared healthy.

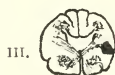
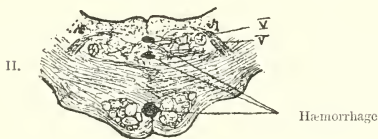
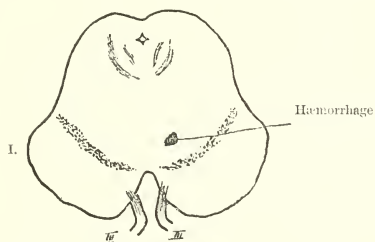
In removing the brain the dura was found to be rather adherent to the skull. There was no flattening of the convolutions, and superficially there was no evidence of disease except the condition of the vessels already referred to. On section of the brain no softening or hæmorrhage was found in the hemispheres, nor could any blocking of vessels be discovered. The corpus callosum seemed normal.

The accompanying drawings which Dr. Colman has been kind enough to make for us show the condition of the parts about the pons. The figures represent the appearance of sections at different levels, between the crura and the spinal cord, and in all of them one or more hæmorrhages can be seen. It is worthy of note that several sections of the pons at intervening points show to the naked eye no sign of disease, the vertical extent of each hæmorrhage being very small.





Section through the pons and cerebellum shewing hæmorrhages into the right middle peduncle.



- I.—Section through the crura cerebri at the level of the third nerve.  
 II.—Section through the pons at the level of the nucleus of the fifth nerve.  
 III.—Section through the medulla at the level of the decussation of the pyramids.

## Clinical Cases.

### A CASE OF BULBAR PARALYSIS WITHOUT STRUCTURAL CHANGES IN THE MEDULLA.

BY LAURISTON E. SHAW, M.D., M.R.C.P.

P. S., æt. thirty-seven, a baker, applied for treatment at the out-patient department at the Victoria Park Hospital, on Friday, February 11th, 1887. From statements he then made, and from facts obtained from his widow on a subsequent occasion, the history of the case is briefly as follows :

He was a married man with seven healthy children. He had always followed his present occupation, that of a baker; he had never suffered from any venereal complaint, and had enjoyed perfect health until his present illness began, six months ago. At that time he was kept from his work by a very severe attack of diarrhœa, which lasted a fortnight, but did not necessitate his staying in bed. Ever since this attack he had suffered from an ill-defined feeling of weakness, becoming gradually more severe, but with occasional remissions. This progressive weakness had been noticed more in the arms than in the legs, and had quite early in the illness been observed in the act of mastication. Throughout there had been wasting of the body generally, and at no time had the patient been well enough to resume his work. During the last month the weakness had been much more marked, and it was within this time that the symptoms had been noticed which led him to seek advice at the hospital.

There had been difficulty of articulation, so that the patient could not make himself clearly understood. At the same time he had found trouble in deglutition—a trouble that was more marked with solid than with liquid food. These symptoms were but slight in the morning, but became more evident as the day advanced. There had been copious secretion of frothy mucus, which he found very difficult to spit out, and which he feared sometimes would choke him. Within the last fortnight he had

two severe attacks of shortness of breath, which gave great alarm to his friends.

He walked into the out-patient room assisted by a friend and required help in undressing. He could however stand and walk a little unaided, but said that he often fell, and was quite unable to walk downstairs alone. When sitting he could not cross one leg over the other without lifting it with his hands. He spoke slowly and indistinctly, reminding one of a patient suffering from tonsillitis. The special difficulty of articulation was not investigated except that he had more difficulty in pronouncing hard "c" than other consonants. There was no nasal twang to his voice, and his soft palate was seen to move well and equally. His mouth was constantly held slightly open, but there was no dribbling of saliva, on the contrary what saliva and mucus collected in the mouth was cleared away with the fingers after futile attempts to expectorate it. There was loss of power in the lower facial muscles, so that the naso-labial fold was obliterated on either side, causing a vacant expression. He was unable to whistle or blow out a light. The loss of power was quite equal on the two sides, and did not affect the upper facial muscles at all, the patient being able to screw his eyes up firmly. The only evidence of any weakness of the muscles of the tongue was a difficulty in starting its protrusion; when protruded it could be moved in any direction, though slowly. The masseters were decidedly weak; the man stating that he was obliged to help mastication by pushing up his jaw with his hand, and complaining that he could not hold his pipe in his mouth either with his lips or his teeth without similar aid. The difficulty in swallowing of which he complained was well shown on his attempting to drink some water. The patient having filled his mouth, threw his head back, when some of the water seemed to get into his œsophagus, but almost at once he choked, and holding his head forward most of it ran from his lips. The laryngoscope showed his vocal cords to move well, whilst the ease with which the examination was made seemed to show some impairment of sensibility of the fauces.

Diplopia had sometimes been noticed by the patient, though at the time of his visit there was none; the pupils, motor oculi muscles and fundus of the eye were all normal.

The muscles of the trunk and extremities were ill-developed and flabby, but there was no obvious wasting of any group of muscles. The grasp was decidedly feeble, but equal on the two sides.

The wrist, elbow and knee jerks were brisk, a little above the average. In the right ankle an attempt to produce clonus resulted in four or five contractions; there was no response on the left side. The cremasteric and plantar reflexes were present and equal; the abdominal and epigastric absent.

His memory and other mental functions appeared normal. Throughout his illness there had been perfect control over his bladder and rectum.

Physical examination of his chest showed nothing abnormal with the heart or lungs. On first lying down for examination the patient complained of difficulty of breathing; no change in the rate of respiration or general condition could be observed, and the sensation soon passed off.

The man became an in-patient on February 12th, 1887. His condition did not attract special attention until the evening of the 13th. At that time he was attacked with sudden and urgent dyspnœa. The resident medical officer who was called to see him considered his condition to be due to paralysis of the intercostals, the expansion of his chest being carried on by the accessory muscles of respiration. He was becoming rapidly cyanosed, so that artificial respiration was tried with good result. The improvement was only temporary, and the proceeding had to be resorted to again and again. The following note was made of his condition in the intervals of the attacks: "His tongue is protruded straight. There are no ocular paralyses; pupils react readily and equally. He speaks clearly. The mouth is not drawn to one side. He cannot swallow his saliva, which collects about the fauces and is spat out with great difficulty, apparently from weakness of the muscles of the cheeks and tongue. Respiration is short and jerky; chest is full of large moist râles. Nothing can be felt at the back of the pharynx. There is no obstruction in the larynx to the entry of air. His hands are cold, and when an attack of dyspnœa is coming on he beats his chest and throws his limbs about in great distress, and asks to have artificial respiration performed. His mind is quite clear."

Subsequently the attacks became more frequent and severe, especially with regard to the urgency of the feeling which the patient described as an inability "to expand his lungs." Artificial respiration was repeatedly performed and for long periods, but this was at last ineffectual and the patient died a few hours after the beginning of the attack.

I made a post-mortem examination of the case on the afternoon of February 14th. Beyond some pus in the smaller

bronchial tubes and an enlarged mediastinal gland, every organ in the body was normal. Especial care was taken to search for signs of old syphilis: there was no scar on the penis and the testes were normal. The vertebral column was healthy, and nothing abnormal found in the upper respiratory passages.

Tonaked-eye examination the brain appeared perfectly healthy, as also did its meninges and those of the spinal cord. The pons, medulla and spinal cord, as being the likely seats of lesion, were handed to Dr. Newton Pitt for microscopical examination. The following is the report of the examination kindly sent to me by Dr. Pitt.

“Pons, medulla and spinal cord normal to naked-eye investigation. Specimen hardened in Muller's fluid, and sections stained some with aniline blue black, some with Weigert's hæmatoxylin stain. Sections have been made and examined from the following situations: through the fourth and first cervical segments, at the lowest part of the medulla, through the medulla just before the canal opens, *i.e.*, at the level of the eleventh and twelfth nuclei, and four others higher up of which one was through the vagal nucleus and one through the locus cœruleus. The cells of the nuclei, the fibres, vessels and all the tissues in every section appear normal, hence any organic disease of these parts of the nervous system may be safely excluded.”

The clinical combination of defects of respiration, deglutition, articulation and mastication, naturally led us to expect the medulla to furnish us with a pathological explanation of the case. The failure to find such explanation adds one more to the many recorded and unrecorded cases in which physiological knowledge and previous pathological experience do not lead to a correct solution of the mystery of disease. Whether this is to be found by examining more carefully the suspected organ or tissue, or by looking elsewhere, or by seeking for possible causes of change in the function of the organ other than visible alterations in its structure can account for, is only to be determined by as far as possible adopting all these methods in every case. The advantage of reporting such negative cases as the one above seems to be that we are kept on our guard against assuming slight structural changes which may possibly be only secondary to the main disease to be its real cause.

# A CASE OF POLIOMYELITIS CHRONICA OCCURRING IN A CHILD AGED FIVE YEARS.

BY F. W. KIRKHAM.

MARY H., a child, aged five years, was brought to me on the 4th September, 1886.

The patient had always enjoyed good health, and had had no previous illness of any description.

She commenced to walk when fourteen months old, her teeth appeared quite regularly. She had never suffered from convulsions.

For about a month before I first saw her she had been noticed to walk somewhat unsteadily, and her legs seemed after a little exertion to be weak and powerless. These symptoms continuing to increase; her parents consulted me about her. When I first saw the patient her appearance and general condition was that of a healthy child.

She was slight but well nourished, and seemed bright and intelligent. On being put upon her feet it was found she could stand with great difficulty, and on walking took hold of surrounding objects to steady herself. There was at that time no apparent wasting of the muscles of the legs or thighs, and the patellar reflex was normal on both sides.

The weakness was stated to be more marked in the left than in the right leg, but, on seating her on her mother's knee, there appeared but little difference between the condition of the two, the movements of both being extremely weak and uncertain.

There was no anæsthesia or paræsthesia, and the powers of micturition and defæcation were unaffected.

On applying the faradic current to the nerve trunks of the lower limbs there was very little response, and the same was noticed of the application of the same current to the muscles. A constant current up to forty Stöhrer's cells applied to the nerve trunks of the lower extremities produced but slight contractions,

a current of twenty cells applied to the muscles, however, produced slow and tonic contractions  $A C C = K C C$ .

I saw the patient occasionally during the next few weeks, but had not an opportunity to test further the electrical reactions till May 14th, 1887. At this time her condition was much worse than when I first saw her, she now being wholly unable to stand or walk, though she could move her legs slightly when sitting. There was also marked atrophy of the muscles of the calves and thighs.

There was no response whatever to very strong faradic currents, whether applied to the muscles or nerves.

There was also no response to the application of a strong galvanic current to the nerve trunks, but vigorous, slow and tonic contractions when a very weak current was applied to the muscles,  $A C C > K C C$ . The patient remained in this condition for about two months, when she was again seen by me, the parents then thinking they observed a slight increase of motor power in the paralysed limbs.

This appeared certainly to be the case, and when the child was again brought to me in November, 1887 there was a very decided improvement, she being able to stand when supported a little. There was now a little reaction when the faradic current was applied to the nerve trunks and muscles of the limbs, and of galvanism to the nerve trunks, but application of a weak constant current to the muscles still produced slow tonic contraction,  $A C C > K C C$ .

I did not see the patient again until the following February, when she had recovered the complete use of her legs, and seemed to be in perfect health.

The electrical reactions were then perfectly normal, and the wasted muscles had increased in volume and were nearly, if not quite, as well developed as in other children of her age.

She has since remained quite well.

## A CASE OF HEMIATROPHY OF THE TONGUE WITH ITS PATHOLOGY.

BY E. F. TREVELYAN, M.D.(LOND.) B.SC.

*Resident Medical Officer and Pathologist, Leeds General Infirmary.*

ANNIE B——, aged twenty-five, was admitted into the Leeds General Infirmary on May 1st, 1889, under the care of Dr. Churton, to whose kindness I am indebted for permission to publish the case. The following clinical notes were taken by Mr. Walker, formerly house physician, and Mr. McCandlish, the clerk of the case:—

The patient, a stout well-built woman, weighing nine stone, complains of pain in the neck, wasting of half the tongue, and chronic abscesses below the jaw. Temperature 99°; pulse 120; respiration 26. The pain in the neck is increased by even slight pressure on the cervical spine; the pain is very severe if the vertex of the skull is pressed upon. When asked to turn her head she turns her body instead, keeping her head fixed. She has some pain in the left ear on swallowing; her hearing is good. She also complains of pain over the left eye. No affection of first, second, third, fourth and sixth cranial nerves. The left half of the tongue is markedly atrophied; it can be moved from side to side both in the mouth and when protruded, but not so easily in the latter case. The left half of the tongue is felt to be thin from wasting of the lingualis. The organ is exactly represented by the drawing in Gower's "Diseases of the Brain" (vol. ii., p. 278). There are marks of former strumous glands on the left side of the neck, two burrowing fistulous sores below the jaw on the right side. The thoracic and abdominal viscera are healthy.

She gave the following history. Seven years ago she had suppurating glands in the neck, but was otherwise quite well up to eight weeks ago. At that time she went out one night and on her return had pain and stiffness in the left side of the neck. This was looked upon as a simple neuralgia, but it did not yield at all to treatment. Three weeks ago she was sent to Har-



rogate, got a chill, and next morning her tongue was swollen, and shortly after this she noticed the left side of the tongue began to waste. Mr. W. H. Brown was called in and recommended her removal into the infirmary. A few days after admission Dr. Churton tested her taste with dry quinine, salt and sugar. There appeared to be *no* taste on the left half of the tongue, either at the top or in the middle, nor even on the palate. The pain in the neck, with inability to move the head or the neck in any direction, continued. She could not wear a support for the head on account of the suppurating glands in the neck. The temperature both morning and evening was high. On June 18th the pain was worse and she began to vomit and to be rather delirious, calling for her father and taking anyone who came to her bedside to be her father, otherwise she was inattentive to what was going on around her. On the same evening she had a fit, foamed at the mouth and bit her tongue. Her face and arms twitched during the fit and she had marked Cheyne-Stokes breathing after it. The face and arms twitched occasionally during the night. Next morning (19th) another fit occurred, the right arm and both sides of the face being convulsed. The pupils were dilated, the face dusky. Temp. 102°. She was quite unconscious. She remained so during the day and died at ten p.m.

The following is an account of the morbid anatomy. The left side of the tongue is much atrophied.

*Occipital Bone*.—In the neighbourhood of both occipito-atloid joints the bone is much roughened and worm eaten. No definite collection of pus was found there.

*Atlas*.—Superior articular surfaces are both diseased, the left being the worse of the two. The cartilage covering them is worn away in parts and distinctly honeycombed in others. This is more marked on the left side. These articular surfaces are less distinctly defined than they should be, a distinct rough surface of bone running round the left one. The bone is also seen to be rough and uneven on the anterior surface of the anterior arch on the left side. There has been clearly a deposit of new bone here. The rest of the bone is healthy. The transverse ligament was found entire.

*Axis* quite healthy except the upper part of the odontoid process, which is roughened. A section taken through odontoid process shows healthy bone. The rest of the cervical spine is healthy.

*Base of Brain*.—There is considerable thickening of the men-

inges at the base of the brain (especially of the pia mater and arachnoid). This is very noticeable about the origin of the left hypoglossal nerve, its two bundles of fibres passing through the thickened membranes. No such thickening can be made out round about the right hypoglossal nerve. There is no tumour of any sort at the base of the brain (nor was there any in the upper part of the neck in the course of the hypoglossal nerve. Patient had suffered from chronic inflammation in the glands of the neck). Sections taken at varying levels through the Medulla Oblongata show no change in the hypoglossal centres. The cells are well developed and no difference is noted between the two sides. No change is visible in the infra-nuclear tract.

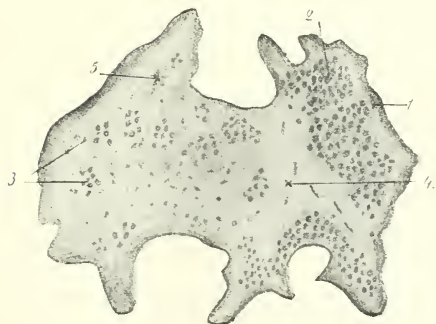
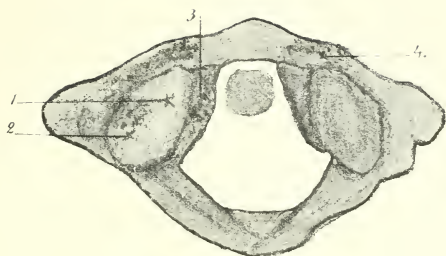
*Nerves.*—Left hypoglossal is undoubtedly smaller and more shrivelled than right. A section treated with osmic acid and stained with *Picro-carmin*e presents the following appearances under the microscope:—The nerve is very irregular in outline, presenting projections and depressions all round its circumference. The epineurium is greatly thickened and bands of connective tissue are seen making their way inwards from the circumference. When the section is examined by a low power fasciculi of nerve fibres are seen scattered irregularly throughout the section. In one place they are more abundant, in another hardly any are to be seen. In between these fasciculi the connective tissue has undergone very considerable increase. It forms a branching net-work enclosing the fasciculi, or what is left of them. It is seen to contain a number of fusiform fibres. Under the high power it may be seen that many fasciculi have perished, leaving hardly any trace of their presence, whilst others are in the process of disappearing. In between the fibres themselves the endoneurium is increased and individual fibres are seen gradually undergoing atrophy.

*Right hypoglossal nerve* is healthy.

*Right and left gustatory nerves* are healthy.

*Left chorda tympani* is quite healthy. This nerve was examined at the instance of Dr. Churton, with relation to the loss of taste on the left side of the tongue.

Hemiatrophy of the tongue is a rare disease, so that it seldom occurs that a case can be reported in its entirety, viz., with an account of the *post-mortem* examination appended. The chief interest of this case lies in the fact that the hemiatrophy was due to one of the rarer causes of this disease (viz., involvement of the nerve in thickened meninges), and that this cause





was made out and verified by *post-mortem* examination. Dr. Jacob, the Honorary Pathologist, examined the body after death, found that basal meningitis was the fatal lesion and that the occipito-atloid articulation with the neighbouring occipital bone was diseased. A more exact description of the affected parts has been given. The history of the case was the usual one of disease of the upper cervical spine—a history of deep-seated pain in the region of the neck, at first supposed to be a simple neuralgia—followed by rigidity of the neck. Five weeks from the beginning of the disease the left half of the tongue began to waste.

On admission into the infirmary she had all the ordinary signs of cervical caries. No other cranial nerves were involved, except in so far as taste was concerned. There was no paralysis of the palate. (Unfortunately a laryngeal examination was not made, but the absence of signs, the site of the disease and the non-involvement of the spinal accessory nerve make it practically certain there was no laryngeal palsy.)

It is proposed here to enumerate the causes of hemiatrophy of the tongue, and to illustrate them from cases which have already been published. We have then to consider the pathological changes which may affect: (1.) The nucleus of origin of the hypoglossal nerve or its fibres as they pass out through the M.O. to emerge between the pyramidal and olivary bodies. (2.) The nerve from where it leaves the medulla in two bundles until it quits the anterior Condylloid foramen as a single trunk, and thirdly and lastly, the trunk of the nerve from the anterior condylloid foramen to its final distribution in the tongue.

(Disease of the hypoglossal path. from its nucleus of origin in the M.O., up to its representative in the cerebral cortex, can hardly give rise to a wasting out of proportion to disease, viz., to such wasting as we are concerned with here.)

(1.) The nucleus of origin and infra-nuclear tract in the M.O. may be affected by (a) hæmorrhage, (b) softening, or (c) degeneration.

(a.) A remarkable case which illustrates atrophy of the tongue as a result of hæmorrhage into the M.O. is related by Dr. Hughlings Jackson (*Lancet*, 1872, p. 771). The atrophy was here curiously bilateral.

The patient, fifty-two years of age, was suddenly seized

while recovering from hemiplegia, with paralysis of the tongue. The tongue subsequently atrophied. There was no laryngeal palsy. *Post-mortem*.—Dr. Lockhart Clarke found among other lesions the remains of a hæmorrhage into the left olivary body.

(b.) The second process that may affect the nerve and its centre in the M.O., is softening. Thus Prof. Hirt (*Berl. Kl. Wochenschr.*, 1883) tells of a patient who was seized with an apoplectic attack. One month later the right half of the tongue began to atrophy. The right recurrent nerve was paralysed. Condition of palate is not mentioned. Taste normal. The lesion was put down as bulbar hæmorrhage with embolic softening of the centres of the hypoglossal and spinal accessory nerves.

(c.) The third morbid change is degeneration. Raymond and Artaud relate (*Arch. de Physiologie*, vol. xvi., pp. 367) a most interesting case. A patient suffering from tabes dorsalis for eight years, began to have difficulty with his speech one year before he came under observation. At this time he had well marked right hemiatrophy of his tongue. Eight years later he died. The cells in the principal nucleus of origin of the right hypoglossal nerve were found degenerate and pigmented.

As regards hemiatrophy of the tongue in tabes dorsalis, M. Ballet has pointed out (*Arch. de Neurologie* vii., pp. 191, 1886) that it may come on early in the disease, that it may be associated or not with ocular palsies or atrophy of some of the muscles of the extremities. This was so in MM. Raymond and Artaud's case, where there was slight wasting of the thenar eminence. More recently Plücker in an inaugural address at Würzburg (*Centralblatt f. Laryngologie u. Rhinologie*, Oct., 1889) reported a case of tabes where left hemiatrophy of the tongue existed early in the disease. Atrophy of half the lower jaw was also present in this case.

This lesion, viz., degeneration may not be limited to the hypoglossal centre, but may involve the neighbouring nucleus of the spinal accessory nerve (the palate being paralysed at the same time), thus giving rise to what has been called associated paralysis. This has nearly always occurred in

syphilitic patients. The exact lesion has apparently hardly ever been proved by *post-mortem* examination, and therefore it cannot be said for certain whether it is a degeneration such as occurs in other parts of the nervous system in syphilitic subjects or whether on the other hand, it is a softening—the result of thrombosis, the latter again being caused by a syphilitic endarteritis. Thus Dr. P. K. Pel relates (*Berl. Kl. Wochenschr.*, 1887) a case of a man, æt. 34, who had left hemiatrophy of the tongue, paralysis of soft palate (left) and left vo. cord. Patient had had syphilis. He had knee jerks, but there were no other tabetic symptoms. Dr. Pel attributes (without *post-mortem* evidence) the disease to softening of the hypoglossal and left vago-accessorius nuclei due to thrombosis of the left anterior spinal artery, a refinement of diagnosis to say the least of it. Several cases of this associated paralysis (half the tongue and palate and the vo. cord of the same side) have been reported from time to time, by Dr. Hughlings Jackson, in the *Brit. Med. Journal*, 1887 (Right). *Lancet*, 1886 (Right), *Lond. Hosp. Reports*, by Dr. S. Mackenzie, *Brit. Med. Journal*, 1883 (left), and *Clin. Soc. Trans.*, 1886. In the second of Dr. Mackenzie's patients there was no history of syphilis, the man attributing his illness to the fumes of Hg., Pb. and Sb., among which he had to work. In some of these cases some other muscles have been paralysed, as for instance, the Trapezius.

Leudet (*Ann. des Mal. de l'Oreille*, Dec. 1887) has also related two cases of associated paralysis with histories of syphilis, none of tabes. Mr. Holthouse reported a case of paralysis of the sixth and ninth nerves in a syphilitic patient. The atrophy (right sided) was hardly apparent in six years time. This case is put in here on account of the affection of the two nerves in a syphilitic subject. The progress of the case and the nerves involved would make it probable that the disease was not of central origin.

Some few cases have been reported of progressive facial hemiatrophy, in which there has been atrophy of the tongue on the same side, for instance Dr. Buzzard's case (*Clin. Soc. Trans.* V.) with right lingual hemiatrophy. Dr. Dreschfeld's case (*Brit. Med. Jour.*, i., 503, 1882) with left hemiatrophy.

Dr. Payne's case (*Brit. Med. Jour.*, 1880, i., p. 743) with left hemiatrophy. On the other hand some cases of this progressive hemiatrophy have been recorded where there has been no atrophy of the tongue. Thus, Prof. Berger (*Deut. Arch., f. Klin. Med. Band.*, xxii., p. 432) tells of a boy who had right hemiatrophy of the face after a fall, in which there was no atrophy of the tongue.

(2.) The hypoglossal nerve may be involved in disease between its exit from the M.O., and where it leaves the anterior condyloid foramen.

(a.) It may be involved in thickened meninges as in the case of Hayem and Girandean (*Révue de Médecine*, 1883). Dr. Churton's case is also a good example of this, the nerve being involved in a thickening of the Meninges which itself was consequent upon disease of the occipito-atloid joint and neighbouring bone.

(b.) The nerve may be pressed upon or embedded in a tumour. In the classical case of Dupuytren, a hydatid cyst had inserted itself into the anterior condyloid foramen. The late Dr. Habershon (*Med. Times*, Feb. 9th, 1881) relates a case where a secondary growth involved the basilar process and extended into the right temporal fossa, involving the eighth and ninth nerves. The sterno-cleido-mastoid muscle was also affected.

A case is reported by ROMBERG (*Nev. D. Syd. Soc. Trans.*, p. 307) where there was probably pressure on both hypoglossal nerves at their roots of insertion into the M.O., this giving rise to complete atrophy of the tongue.

Mr. FAIRLIE CLARKE reported a case of hemiatrophy of the tongue, which was caused by a secondary tumour within the brain or high up in the neck (*Med. Chi. Trans.*, vol. vii., No. 1, 1872.)

A most interesting case has been reported by LEWIN (*Jahres. Gesamt. Med.*, 1883) where the hypoglossal nerve (left) was involved in a gumma which was seated in front of and in the anterior condylar foramen. The patient had left lingual hemiatrophy. There was diminished tactile sensation and sensation to heat and pain. Taste perceptions lessened. There was salivation. There were also gum-



mata at the root of the tongue. Nucleus of hypoglossal nerve healthy.

(c.) The nerve may be involved as it passes through the anterior condyloid foramen by thickening or cancerous disease of the bone.

A very interesting case is related by Dr. HUGHES BENNETT, where the whole tongue was atrophied, but the case is so important that it can hardly be passed over in any account of hemiatrophy of the tongue. Patient, æt. 54, had a tumour removed from the breast; eight years later she had atrophy of the tongue. She had peripheral disease of the facial nerves as well as of the hypoglossal. At the *post-mortem* examination numerous patches of degenerated bone due to malignant disease were found within the cranium, varying from a pea to a shilling in size. On careful examination the internal auditory meatus and anterior condyloid foramen were involved on each side, in such a way as to cause pressure on the nerves and peripheral degeneration. The symptoms resembled bulbar palsy in life, the reaction of degeneration being one of the chief distinguishing points.

(3.) The nerve may be damaged in its course from the anterior condylar foramen to its final distribution in the tongue, by (a) injury, or (b) disease.

(a.) Dr. HYDE SALTER (*Encyclop. of Anatomy and Phys. Art. Tongue*), relates the case of a man who was stabbed in the neck. Both hypoglossal nerve and carotid artery were divided. Left hemiatrophy followed. Bernhardt (*Deut. Archiv. f. Klin. Med. Band. xxii., S. 392*), that of an attempted suicide who divided his hypoglossal nerve.

WEIR MITCHELL (*Injuries to Nerves*, p. 335), that of a lad who had a gunshot wound which injured the hypoglossal nerve, hemiatrophy of the tongue followed.

Much more recently Mr. RUTHERFORD MORRISON (*Brit. Med. Jour.*, 1888), has put on record the case of a girl who strained her neck in jumping over a grave. Paralysis (followed by atrophy) of the right half of the tongue ensued. Morrison attributed the disease to a hæmorrhage into the sheath of the hypoglossal nerve or else to a stretching of the nerve over the long process of the atlas.

Sir JAMES PAGET, long ago in the *Clin. Soc. Transactions*,

related a most interesting case of a young man who fell heavily upon his head. Paralysis of the right side of the tongue with wasting followed. An abscess formed and discharged. Dead bone was removed and the man recovered.

ERB, in his article on paralysis of the hypoglossal nerve (in the *Arch. f. Klin. Med.* xxxvii., p. 270) mentions that cases of paralysis with wasting have occurred as a result of dislocation of the atlas. These have been related by Uhde Hagemann and Böttche in Langenbeck's *Arch. f. Chirurgie*.

(b.) Hemiatrophy of the tongue may be caused by the nerve being involved outside the skull in growths, or even in enlarged glands, as related by Mr. Hutchinson (*Med. Times*, 1880, vol. 1. p. 57) in a case of malignant growth, and by Prof. Bernhardt (*Arch. f. Klin. Med.* B. xxii., S. 392) in a case of glandular enlargement.

Again, there seems to be no reason why the hypoglossal nerve should not be the seat of a neuritis, and this in turn, be followed by lingual hemiatrophy. Erb in the place cited above, relates a case of the disease of the hypoglossal nerve. After a full and admirable discussion he comes to the conclusion that the case was one of peripheral neuritis of rheumatic origin.

It may be well to conclude this paper by relating a case of great interest recorded by REMAK (*Centralblatt f. d. Med. Wissensch.*, 1886) in which there was a right hemiatrophy of the tongue in a case of lead poisoning.

Patient, æt. 43, a worker in lead, and addicted to alcohol had right wrist-drop and right hemiatrophy of the tongue. Pupils did not act to light. There was some limitation of ocular movements to the right. Paralysis of right post crico aryt. M. and Thyro arytenoid M. Knee jerks present, no signs of tabes.

The presence of Pb. poisoning is certainly suggestive that the lesion might be of peripheral origin, but again, the fact of its being associated with paralysis of the right cord receives an easier explanation if it be supposed to be of central origin. The close approximation of the centres of the hypoglossal and spinal accessory nerves easily explains the fact that a morbid process might spread from one nucleus to the other, and this appears to be a less strained explanation than one which supposed these two nerves (not at all susceptible to inflammatory change) to be independently the seat of a neuritis.

REMAK states that he believes the disease to be of central origin, and the excuse for introducing the case here must be that the behaviour of cases of lead poisoning is usually such as to suggest a peripheral rather than a central lesion.

## CASE OF GENERAL PARALYSIS COMPLICATED BY APHASIA.

BY C. PRICE TANNER.

*Assistant Medical Officer, Powick County and City Asylum, Worcester.*

I AM indebted to Dr. E. Marriott Cooke for permission to publish the notes of the following case :—

T. R., æt. thirty-three, married, telegraph clerk, was admitted into the Worcester County and City Asylum September 19th, 1889. The history obtained from his wife was to the effect that he had always enjoyed good health, and had had no illness till twelve months before admission. At that time he suffered from “writer’s cramp,” was low spirited, and seemed generally ill and out of sorts. He went for change of air to Lancashire, and after two or three months had apparently quite recovered. He remained well till August 24th. On that day, about 9.30 a.m., he was about to read the paper aloud to his wife, when he suddenly began to stammer and “talk jargon.” He was put to bed, and soon afterwards his right arm and leg were found to be paralysed, and his mouth seemed to be drawn to one side. He was unconscious, and remained in this state till two a.m., August 28th, when he began to try to talk. His language, however, was quite incomprehensible. From that date till the time of his admission the paralysis has gradually improved, the improvement starting in the leg before the arm, but his speech has never become more intelligible, though he can utter more words now than he could at first. After being in bed about a fortnight he got up, and then was noticed an entire change in his moral character. From being a fond father he seemed to take an aversion to his children, and was found by his wife more than once ill-using them. On one occasion he was holding one of them on the floor, kneeling over her with a knife in his hand. He also became very suspicious, and since his attack has always slept with a hammer and knife by his bedside. He has also lately had extra locks and bolts put on his doors. In consequence of his threatening behaviour to his wife

and children it was found necessary to put him under restraint. He has had five children, all of whom are alive and healthy. There is nothing in the family history bearing upon the case in any way.

In the certificate upon which he was admitted into the asylum, it was stated that he was constantly muttering to himself, wandered about in an aimless manner, had threatened his wife with a poker, imagined he was telegraphing up the chimney and listened for a reply. He was stated to be epileptic on the strength of the "fit" of August 24th. *On admission*, he was found to be thin and poorly nourished, but free from bruises or marks of injury. All his thoracic and abdominal organs seemed to be acting healthily. There was slight tremor of the facial muscles; pupils equal; tongue deviated a little to the left side. The right limbs were weaker than the left, showing signs of the late hemiplegia. No wasting of muscles. Speech very aphasic (*vide infra*). Mentally he appeared to be in a condition of mania. Was extremely restless and agitated, wandering up and down the ward in an aimless manner, chattering to himself, and constantly trying to undress himself.

*September 25th.*—His aphasic symptoms are as follows:—He is quite unable to understand spoken words, except when asked his name. Cannot read printing aloud, though he can tell when the book is held the wrong way up. Cannot repeat spoken words, write at dictation, or copy printed matter. Can understand written words to a fair extent, and read them aloud; but in doing so makes mistakes in the pronunciation of letters, *e.g.*, calls "name," "tame." In copying writing frequently transposes the letters, but puts them all in, copying in this way even words spelt wrong, *e.g.*, he copied pensil as "pensli." He recognizes objects by sight, but apparently not by hearing or smell (touch?). He can articulate, but uses wrong letters, calling knife, "life," &c. The time 4.40 he calls "forty—forty-one—one." Can write his name correctly, but cannot pronounce it. On being asked (in writing) his age, he wrote down "about nineteen years of age." After careful testing no hemianopsia could be discovered.

*October 10th.*—At times seems very much frightened when spoken to, and on one occasion recently began to cry when given a pencil. His aphasia has slightly improved, and he can utter a few sentences and write a few words. His tongue has been noticed to be very tremulous when protruded. Has been put on a mixture containing hyd. perchlor. and pot. iod. tentatively.

*November 3rd.*—His aphasic symptoms vary a good deal from

day to day ; sometimes he will write a few words, but rarely. At times appears very much frightened and distressed. He knows his bed and his place at the table, &c., and makes himself useful about the ward to a limited extent. Is very clean in his habits.

*November 23rd.*—He appears to be more demented. He does not realise where he is, and seems lost generally. He will sit still in one place for hours at a time doing nothing, and making no effort to amuse or occupy himself. Constantly makes a humming noise to himself, and if made to desist starts again almost immediately afterwards. When his wife or children are mentioned to him (in writing) he does not seem to take the slightest interest in them.

*December 2nd.*—On the 28th ult. he had a succession of thirteen fits in about five hours. He was apparently dying, and was with difficulty revived by enemata of brandy and chloral hydrate. The fits began in either one or the other hand, and then spread to the whole body ; the head and eyes rolled slowly from side to side, generally to the left first. He is now confined to bed ; both legs are completely paralysed and he is passing all his dejecta into the bed. A bedsore is beginning to form over the right trochanter. The knee-jerks, which were exaggerated after the fits, are now absent. Slight clonus of right ankle.

*December 4th.*—Both legs are becoming contracted at the hips and knees. Knee-jerks and ankle clonus entirely absent.

*December 7th.*—In spite of everything which could be done, the bedsore has spread in a most malignant way, and to-day a large slough has separated, leaving a deep unhealthy ulcer, almost exposing the femur.

*December 10th.*—Sores forming over left hip and sacrum ; tongue dry, coated. Patient tries to pull off the dressings.

*December 11th.*—Sores beginning to form over both heels.

*December 17th.*—To-day began to refuse food. Is coaxed to take beef tea, milk and brandy. Restless, pulling off his dressings unless closely watched.

*December 31st.*—Since last note has grown gradually weaker ; the sores have increased in area and depth. For the last two or three days has only been kept alive by the frequent administration of brandy and milk in small quantities. He died this evening.

---

Permission could only be obtained for an examination of the brain.

*Head.*—Skull-cap regular, of medium thickness and density.

Dura mater not unduly adherent to bone. No surface hæmorrhages. Arachnoid and pia mater thickened, opaque and milky. Large quantity of fluid in meshes of membranes. Adhesion and decortication well marked over both convexities, principally over the ascending motor convolutions of the two sides and the frontal convolutions of the left side. Brain generally wasted; convolutions flattened externally. White matter pale and firm to the knife. Grey cortical matter thin and anæmic. Ventricles enormously dilated, full of clear, pale, serous fluid. The amount of fluid that escaped after removal of the brain was at least seven ounces. Frosting present in lateral ventricles. Foramen of Monro dilated to the size of a threepenny piece. Centres of right side appeared healthy; those of left side compressed laterally. The whole of the left insula was destroyed, apparently the result of a hæmorrhage, its place being occupied by a mass of broken-down blood-clot, apparently becoming encysted. The hæmorrhage had invaded the first temporo-sphenoidal lobe near its tip, and also the part of the inferior parietal lobe, where it is adjacent to the fissure of Sylvius. This was the only point at which the lesion appeared on the cortex of the hemisphere. The surrounding parts were stained with blood pigment. Extending internally, the lesion had destroyed the claustrum, whilst the external capsule, lenticular nucleus and internal capsule were flattened in the direction of the lateral ventricle. The source of the hæmorrhage could not be traced. No other gross lesion existed. The cerebellum, pons and medulla oblongata were normal to the naked eye. No frosting in the fourth ventricle. Slight atheroma of the arteries at the base of the brain.

*Weight of Brain.*—Immediately after removal,  $48\frac{1}{2}$  ounces; stripped of its membranes and free from fluid,  $36\frac{3}{4}$  ounces.

*Remarks.*—The above case presents, I think, features of interest, both on account of the obscurity of some of the symptoms, and the difficulties attending a correct diagnosis. The history clearly pointed to a cerebral lesion in the left hemisphere, probably cortical or sub-cortical in situation. Of the cause of this lesion, beyond the fact that it was almost to a certainty vascular, no evidence could be obtained. The fact of the paralysis clearing up in the limbs whilst the aphasia remained would limit the situation of the lesion to the neighbourhood of the convolution of Broca, that centre having been permanently injured or destroyed, whilst the centres for the limbs had been merely inhibited. The

aphasia was both motor and sensory—the latter including almost total word-deafness and word-blindness to a slighter extent. The co-existence of word-deafness with word-blindness would indicate lesion of the temporo-parietal region, as explained in a paper by Dr. Allan Starr, of New York, in a recent number of *BRAIN*;<sup>1</sup> and *post-mortem* appearances justify that conclusion in the present case, as the first temporo-sphenoidal convolution and—to a less extent—the inferior parietal lobule were involved in the lesion. The absence of hemianopsia would exclude the angular gyrus from participating in the lesion, though its association tracts must have been damaged, as was proved by his inability to read aloud. The loss of power to recall the names of recognised objects indicates, in a similar manner, lesion of the association tract between the occipital and temporal areas, and as the inferior parietal lobule was involved, this case also agrees with Dr. Starr's statement.<sup>2</sup>

Another point of interest is the extreme rapidity with which the symptoms of general paralysis, when once established, ran their fatal course. From the date of admission to the patient's death was barely fifteen weeks, and for the first three or four weeks of that time, at least, the symptoms were at best equivocal. Though his case was looked on with suspicion from the time of admission as possibly one of general paralysis, it was impossible to point to any group of symptoms that would indicate that condition with certainty.

Apart from the physical symptoms, however, this case also presented unusual difficulties with regard to the mental state of the patient shortly after admission, which led to some amount of correspondence with the Commissioners in Lunacy. Judging from the previous history, the man was undoubtedly a proper person to be put under care and control and detained in an asylum, yet for some weeks after his admission (after he had once settled down in his new quarters) his insanity was by no means obvious, and not until the bodily symptoms left no doubt as to the grave

<sup>1</sup> *BRAIN* July, 1889, p. 95.

<sup>2</sup> *BRAIN*, July, 1889, p. 92.

nature of his malady, did his mental state—as exhibited in the asylum, and judged apart from the previous history—warrant a diagnosis of insanity.

I can only regret that a more complete examination *post-mortem* could not be made. The fulminating character of the trophic changes might have been explained by an inspection of the spinal cord, and an examination of the other viscera might have cleared up the mystery surrounding the origin of the cerebral lesion.



## Critical Digests.

### CHRONIC HYDROCEPHALUS.\*

BY M. ARMAND RUFFER, M.A., M.D.

MOST historical sketches of diseases begin by a reference to the father of medicine, Hippocrates, and by a quotation of his views on the subject to be discussed. Strangely enough this author, who has given graphic sketches of many strange and rare diseases, does not mention hydrocephalus in any of his works. After a search in the original, with the help of Littré's<sup>1</sup> translation, no reference to it could be found. It is true that, writing of epilepsy in the sixth volume of his works, he says<sup>2</sup>—"For when the disease has lasted long it is curable no longer, the brain being eaten away by the fluid and melts; the part thus melted is converted into water, which surrounds the brain and bathes it, causing the attacks to become more frequent and more easy," but it is doubtful whether this refers to chronic hydrocephalus or not. The lines in vol. vii. p. 27,<sup>3</sup> in which he talks of water forming on the brain, evidently refer to meningitis. It is impossible therefore to say for certain whether Hippocrates knew anything about chronic hydrocephalus.

Galen,<sup>4</sup> (Book xiv., page 782) states that :—"There are four kinds of hydrocephalus : the first between the brain and meninges ; the second between the meninges and the bone ; the third between the bone and the pericranium ; the fourth between the bone and the skin. The hydrocephalus between the skin and the pericranium we treat with two or three free incisions ; that between the meninges and the brain is incurable." It is not possible to say whether "*τὰ μὲν μείυξυ*

\* Abstract of Thesis written to obtain the Degree of Doctor of Medicine of the University of Oxford, June, 1888.

*ἐγκέφαλον καὶς μήνιγγος*” refers to water contained in the brain itself or present between the brain and the membranes covering it. In any case his knowledge of the subject was extremely vague.

Celsus<sup>5</sup> gives the following account of hydrocephalus:—  
 “Praeter haec etiamnum invenitur genus quod potest longum esse ubi humor cutem inflat, eamque intumescit et prementi digito cedit: *ὑδροκέφαλον* Groeci appellant. “Besides these there is also found a kind which may be of long duration, and where the fluid swells up the skin, raises it and gives way under the pressure of the finger, the Greeks call it hydrocephalus.”

Oribasius,<sup>6</sup> living in the 4th century, says that:—“The disease called hydrocephalus is the result of the compression of the head by midwives during the birth of the children. The patients afflicted with this disease present on the head a considerable tumour, elongated in form, giving rise to no little disfigurement. It is impossible to have an accumulation between this membrane (meninges) and the brain itself, for an individual suffering in this way would die before the disease called hydrocephalus had time to develop itself!” The first part of this quotation evidently refers to cephalo-hæmatomata, whilst the second sentence shows that Oribasius was ignorant of the existence of the disease now called hydrocephalus. Further on, he adds that “When the liquid is accumulated between the membrane of the brain and the bone, there will undoubtedly be a tumour, for in young children the bone not yet being ossified moves about, is easily depressed, and hence is easily distended by the pressure of the fluid; but one may see, at least, a solid painful tumour, which elongates into a sharp point. One finds the regions of the forehead and temples thinner than would be expected, having regard to the development of the body, for the parts in this region become contracted, being drawn upwards. These patients have flashes before the eyes and blinking of the eyelids!” The first part of this second quotation may possibly refer to meningo-encephalocèles, and the last shows that he was aware of the existence of nervous symptoms in these patients.

Hildanus de Hilden<sup>7</sup> relates three cases of the disease, and Vesalius<sup>8</sup> has given an account of one. Their accounts refer to young children, whose heads slowly and gradually filled with water, became of monstrous volume, the increase being due not only to widening of sutures, but also to the undue development and widening of the bones of the cranium. This last mode of growth was, in Vesalius's opinion, sufficient to account for the widening of the head in one case; the cranium acquired the circumference of two feet and ten inches in the course of fifteen years. In the three cases of Fabricius<sup>9</sup> the *post-mortem* examination showed that the head had been converted into a membranous cyst, which gave issue to a large quantity of water, amounting to eighteen pounds in one patient.

This subject could not escape the acute power of observation of Ambroise Paré.<sup>10</sup> In his works he first gives an account of hydrocephalus outside the cavity of the skull, attributing to traumatism a large share in its production, and after discussing Vesalius's case of true hydrocephalus, he goes on to say:—"Or, lors que cette humeur est entre le crâne et le dure-mère, ou aux ventricules du cerveau, et en toute la substance, le malade a les sens hébétés, comme l'ouye et la veue, et quand on presse sur la tumeur, elle ne s'enfonce, pas aisément . . . . Albucasis escrit avoir veu un enfant duquel la teste s'agrandissant tous les jours par l'aquosité contenue dedans, enfin devint si grosse, qu'il ne la pouvait soutenir debout, ny assis, et mourut quelque temps après. J'ai veu quatre enfants malades de cette affection de la teste, de l'un desquels je fis section après sa mort, et proteste n'avoir trouvé de cerveau non plus gros qu'un esteuf, et jamais n'en ai veu un seul guérir, lorsque l'aquosité est en grande abondance aux parties intérieures de la tête, mais lors qu'elles est aux parties extérieures on reçoit guérison. Donc si l'humeur est seulement entre le cuir musculoux et le péricrane ou entre le péricrane et crâne, si elle est petite faut essayer à la résoudre, et si l'on ne peut il faut faire incision, et fait sortir l'humeur, évitant le muscle temporal et faire sortir l'humeur, qu'on trouve comme une laveure de chaire sanglante : autrefois de sang noir : et si elle est causée de contusion, on trouve

avec le dit humeur du thrombus de sang. Ayant fait l'ouverture, et l'humeur étant évacué, on remplira la playe de charpie sec, et sera mis par dessus une compresse faisant ligature propre ; et le reste de la cure se parfera comme il sera nécessaire."

Morgagni<sup>11</sup> in his classical work, *De sedibus et Causis Morborum*," gives the result of several autopsies of cases of hydrocephalus as well as of some *post-mortem* examinations on congenital absence of the brain and spinal cord, encephaloceles and spina bifida. He, at the same time, mentions several authors who had written on the subject. This chapter is one of the most interesting in this classical work, for after most accurate descriptions of the pathological specimens, Morgagni bases on these facts theoretical considerations to explain their occurrence. Among the authors quoted by Morgagni are Hunauldus, Hildanus, Paisenius, Pechlinus, Stalpartius and Brinius.

The following are among the interesting cases mentioned by Morgagni. In the first, diminution of the hydrocephalic head followed on tapping a spina bifida, whilst, in the other, the cure of a spina bifida was followed by an increase in the size of the head. In another part the same author states that it does not follow that because a patient has a spina bifida, hydrops of the head must necessarily be present also. This statement shows that even in his time, the existence of a relation between the two diseases was an established fact. We may conclude therefore that in the 16th and 17th centuries, the existence of a disease corresponding to what is now called chronic hydrocephalus was undoubtedly established.

Yet neither Ambroise Paré, the greatest accoucheur of his time, nor, according to Poulet, any of the authors before the 18th century, mention hydrocephalus as a cause of complication in labour.

Wepfer<sup>12</sup> of Schaffhausen in 1665, saw two cases of foetal hydrocephalus giving rise to dystocia, but unfortunately his paper on the subject was not published till 1727, long after his death. In the first edition of De la Motte's<sup>13</sup> works there is an account of two cases of undoubted hydrocephalus

causing dystocia, one being a head and the other a breech presentation. Smellie,<sup>14</sup> however, was the first to attract, in 1752, universal attention to the subject. He not only published several cases but also perforated the foetal head in one case and laid down definite rules for the guidance of the accoucheur. Solomon Naumann<sup>15</sup> (1762) and Murray<sup>16</sup> (1797) wrote elaborate treatises on the subject, whilst Camper carefully studied its pathology in 1785, and Baudelocque<sup>17</sup> gave valuable indications as to diagnosis, prognosis and treatment. Since that time various observers, including Simpson,<sup>18</sup> Cazeaux,<sup>19</sup> Tarnier,<sup>20</sup> Depaul<sup>21</sup> and especially Chassinat<sup>22</sup> have discussed the subject in extensive papers. Lately, the most important works have been those of Ramsbotham<sup>23</sup> in 1867, McDonald<sup>24</sup> in 1878, Fritsch<sup>25</sup> in 1876 and Klebs<sup>26</sup> in the same year. In most text-books on midwifery, especially those of Spiegelberg, Schröder, Lusk, and Charpentier, a good account is given of the disease in its relations to midwifery.

The last paper, that of Pouillet,<sup>27</sup> written in 1880, is perhaps the best sketch of the disease in its relations to midwifery that has been written up to the present time.

But although the disease has been studied by many observers, the pathology of this affection is still enveloped in many obscurities. Tulpius,<sup>28</sup> Wepfer,<sup>29</sup> Bonnet,<sup>30</sup> Val-salva,<sup>31</sup> Morgagni,<sup>32</sup> Lientaud,<sup>33</sup> give *post-mortem* records of hydrocephalic patients, but the mode of increase in the size of the skull and the ætiology of the disease are hardly mentioned. J. L. Petit, in 1718, Robert Whytt,<sup>34</sup> as well as John Warren<sup>35</sup> in 1788, added to the knowledge already gained and proved the existence of a chronic form of the disease in contradiction to Fothergill's<sup>36</sup> opinion, who believed in the existence of the acute form only. Morgagni had, however, before them fully discussed the pathology of the disease. These authors agree in finding the dura mater more adherent than usual, the base of the brain flattened and the ventricles so much distended that the grey and white substance of the brain measure but a few lines in thickness.

More lately, especially since the discovery of the cerebro-

spinal fluid by Magendie and of the foramen bearing his name, the pathological appearances have been assumed by some to be the result of an obstruction to the free flow of the cerebro-spinal fluid. Hilton<sup>37</sup> seems to have been the first to propagate this idea. It was then taken up by Archambault and others, and mentioned in all the text-books of pathological anatomy. Although an obstruction undoubtedly exists in many cases, it is, in the writer's opinion, frequently absent, or, on the other hand, may be present without occasioning hydrocephalus.

During the present century the disease has found a place in all text-books of pathology (Virchow, Lancereaux, Cornil et Ranvier, Ziegler, &c.), but little that is new has been discovered. Kundrat's<sup>38</sup> paper is a most valuable contribution to the study of one form of the disease, but most records of cases and *post-mortem* examinations are found scattered in the transactions of many medical societies.

In this paper the writer hopes to have made use of nearly all the literature of the subject, and to give a fairly full account of what is known of this affection.

## I.

The brain in the embryo<sup>39</sup> presents itself in the anterior dilated portion of the primitive medullary tube, and forms the three primary cerebral vesicles. These primary cerebral vesicles are named fore-brain, mid-brain, and hind-brain, and correspond most nearly to the regions of the *third ventricle*, the *corpora quadrigemina* and the *medulla oblongata* of the adult brain. The changes which mainly tend to modify the form of the primitive brain are:—

1st. The development on each side from the anterior vesicle of the primitive ocular vesicle.

2nd. The protrusion somewhat later from the fore part of the anterior cerebral vesicle of a bulging part, at first single or undivided, but which, by a median cleft and lateral expansion, becomes later the rudiment of the true cerebral hemispheres.

3rd. The formation in the fore part of the posterior vesicle of a new encephalic rudiment corresponding to a cerebellum. Thus, the first vesicle becomes converted into

the cerebral hemispheres and vesicle of the third ventricle or thalamencephalon; the middle vesicle remains undivided and the hinder vesicle becomes the cerebellum and medulla oblongata.

There are thus distinguished the rudiments of five fundamental constituents of the adult brain.

1st. The cerebral hemispheres with their ventricular hollows or lateral ventricles, the *corpora striata* and the olfactory lobes—a set of parts to which, as a whole, the name of pro-cerebrum or pro-encephalon may be given.

2nd. The thalamencephalon with its cavity or third ventricle, the primary ocular pedicles and the infundibulum.

3rd. The mesencephalon comprising the corpora quadrigemina and crura cerebri with its contracted internal hollow—the *iter a tertio ad quartum ventriculum* of human anatomy.

4th. The next part in succession is the cerebellum, along with which is included the pons Varolii and part of the fourth ventricle.

5th. The hinder part, which passes into the spinal marrow, is the *medulla oblongata*, with the remainder of the fourth ventricle and its continuation into the central spinal canal.

In the adult the brain contains five cavities, which are all in communication, namely: (1) the two lateral ventricles, communicating by the foramen of Monro with the (2) third ventricle, communicating through the *iter a tertio* with the (3) fourth ventricle, which itself communicates with the spinal canal by the foramen of Magendie.

Further, the ventricles of the brain communicate with the sub-arachnoid space by means of the foramen of Magendie, the opening into the lower part of the fourth ventricle, through the expansion of the pia mater (*tela choroidea inferior*) which closes it. Two other openings through the membrane exist, one on each side behind the upper roots of the glosso-pharyngeal nerve, in the pouch-like extension of the membrane beneath the flocculus.

A certain quantity of the fluid is contained between the arachnoid membrane and the dura-mater; but the larger



quantity of the cerebro-spinal fluid is lodged in the sub-arachnoid space in the meshes of the trabecular tissue. It must be remembered that fluid injected into the sub-arachnoid space passes freely into the Pacchionian bodies, is found after a time to filter through their walls and thus to get into the sub-dural space, although there does not appear to be any open communication between the interior of these bodies and the prolongation of the subdural space which surrounds them. Moreover, if the injection be continued, it can be driven even into the interior of the venous sinuses and lacunæ which are found in connection with them, especially into the superior longitudinal sinus, into which the arachnoidal villi project. Thus these villi seem to afford a means of passage of the cerebro-spinal fluid from the sub-arachnoid space into the venous sinuses, when the fluid pressure in the sub-arachnoid space becomes from any cause increased above the normal.

Lastly, Axel Key and Retzius have been able to inject the ventricles of the brain by introducing finely-divided coloured fluids into the sub-arachnoid space.

Hence collateral channels are provided by nature for the cerebro-spinal fluid to escape in case of closure of Magendie's foramen. Whether the collateral circulation in case of closure of the latter foramen ever becomes quite adequate to the extra work, remains as yet an open question.

Whilst reading through the literature of the subject the writer met with the records of a number of cases in which only part of the brain is converted into a kind of cyst, this pathological cavity often, though not always, communicating with the ventricles (*porocephalus*).

It was his intention at first to exclude all such cases, but on further consideration he found it impossible to do so, as, for reasons to be stated later on, he came to the conclusion that there was no rigid line of demarcation between *porocephalus* and *chronic hydrocephalus*. In this paper, therefore, the two conditions will be discussed together, although for the sake of clearness the pathological anatomy and clinical symptoms of each will be spoken of separately.



The localisations of each disease are first described; secondly, the morbid changes in the walls of the cavity containing the fluid and in the vessels of that part; and, lastly, the changes in the skull and other parts of the body will be studied.

In the medical literature of this century the records of *post-mortem* examinations in which *localised* collections of fluid in the brain have been found are but few, and the writer, after a careful search, has been unable to collect more than 114 cases of this rare affection.

In 30 per cent. of these both sides of the brain were almost completely destroyed by the contained fluid, and in 10 per cent. the whole of one hemisphere was similarly affected. No marked disproportion existed between the number of cases in which the one or the other hemisphere was the seat of disease. In 52 per cent. the lesion was limited to part of one hemisphere.

In 15 per cent. of these the anterior portion of the brain was the seat of the disease, in 61 per cent. the middle part, and in 16 per cent. the posterior lobes were affected. In 5 per cent. the lesion was situated in both the anterior and middle part, and in 1 per cent. in the cerebellum.

Kundrat's results differ slightly from these. This author states that in twenty-seven cases the seat of the disease was found in the parts supplied by the middle cerebral arteries, in three in the region of the anterior cerebral and in five cases in that of the posterior cerebral.

In 8.7 per cent. of cases tabulated by the writer, one horn of one ventricle only was dilated, the dilatation being most frequently present in the lateral and in the fourth ventricles (no cases of meningocele or encephalocele are included in these statistics). Moreover, a few cases have been excluded in which the lesions were very small and bilateral, or recorded with insufficient minuteness to admit of classification.

The cavity containing fluid communicates with the lateral ventricles in a large number of patients, and this is the case not only when both or the whole of one hemisphere having been destroyed, the brain or one side of it is con-

verted into one huge cavity containing fluid, but also in the slighter forms of destruction.

In 46 per cent. it is distinctly noted that the fluid passed with the greatest ease from the cyst into the lateral ventricles, or *vice versâ*. In the other accounts of *post-mortem* examinations no mention whatsoever is made as to the presence of a communication between the cyst and the normal cavities existing in the brain. The following cases have been selected among many others as typical examples of some of the pathological appearances found in these patients.

In a child<sup>40</sup> born at full term, the surface of the cranium was covered by a red membrane, stretched over a mere crust of bone. On removing this crust some serum escaped. Both cerebral hemispheres and the cerebellum were absent. In another patient<sup>41</sup> the roof of the skull was lined by a vascular membrane mottled with dark red spots like an old apoplectic cavity, another very dense and brownish-red membrane covering the base of the brain. The base itself consisted of the following structures :

(a) Corpora striata, brownish red in colour, indurated and unequal in size.

(b) Optic thalamus.

(c) Some atrophied convolutions at the base of the brain. The olfactory nerves were atrophied, and only traces of the optic commissures and anterior pyramids were found, but the olivary bodies, tubercula quadrigemina, pons and cerebellar peduncles were healthy. Nothing remained of the convexity of the brain, in another case, but a thin greyish layer applied to the falx cerebri and a few traces of convolutions at the base. Cruveilhier<sup>42</sup> has also described another specimen, in which the cortex of the cerebral hemispheres was replaced by a thin brownish membrane covered with granulations. The convolutions were represented by a few very small and dense little masses, forming irregular cylinders curved on themselves. Other noticeable points were the complete absence of the pyramids and large development of the olivary bodies. The cerebellum and cerebellar peduncles were quite healthy, whilst the optic nerves were atrophied and the olfactory nerves absent.

Schwarz<sup>43</sup> could not find a trace of cerebral hemispheres in a patient whose pons, medulla and cerebellum remained intact, the superior sympathetic ganglion being slightly hypertrophied.

The pons, medulla, cerebral peduncles, corpora striata, and optic thalami were quite normal in a specimen shown by Budin<sup>44</sup> to the Société Anatomique of Paris, but the two cerebral hemispheres were represented by the temporal and occipital lobes only, which extended to the internal occipital fissure. Duret,<sup>45</sup> who examined this brain, noticed that the distribution of the lesion corresponded to the areas supplied by the anterior cerebral and sylvian arteries. The carotids were small, and the sylvian and anterior cerebral arteries resembled those of a fœtus. He thought, therefore, that a disease of the membranes (congestion, hæmorrhage) at some period of foetal life (four months) had been the cause of the malformation of the hemispheres. A fact in favour of this view was that the internal aspect of the pia mater over the anterior lobes had a rusty tint like that found in the site of an old hæmorrhage. Dickson<sup>46</sup> recorded a very similar case in 1838.

In a patient whose case has been reported by Binswanger<sup>47</sup> there was a complete atrophy of the third left frontal convolution, of the two inferior thirds of the ascending parietal and ascending frontal convolutions; the angular gyrus, first temporal and lobule of the insula being almost wholly destroyed. The atrophy was therefore almost limited to the parts supplied by the sylvian artery, this vessel not being discoverable. The ventricle on the same side was dilated, but the right hemisphere had not undergone any compensatory enlargement. In a girl, twenty-six years old, Frigario<sup>48</sup> found at the level of the right parietal lobe a sac containing about eighty grammes of yellow and transparent liquid, the smooth walls of the cyst being continuous with the dura mater. The choroid plexus lay at the bottom of the sac, together with one extremity of the right optic thalamus. The cerebral lesions affected the right motor region, the ascending parietal, superior and inferior parietal lobules, part of the angular gyrus and superior part of the quadrate lobe.

On the left side the circumvolutions of the quadrate lobe were rudimentary. An interesting case published by Dr. Cunningham<sup>49</sup> in the thirteenth volume of the *Journal of Anatomy* (p. 508) may also be included in this category.

The walls of the cyst have not always been carefully described. Schwartz,<sup>50</sup> however, mentions that the membranes covering the cavity were formed by an outer layer—the dura mater—and an inner very thin and fragile layer, which appeared to be the ependyma.

According to Kundrat,<sup>51</sup> the meninges are usually healthy. Cotard<sup>52</sup> describes the meninges as being bound more or less extensively to the ventricular membrane. Vessels run between these two membranes and near the boundaries of the cavity, the wrinkled convolutions being transformed into a gelatinous substance before they wholly disappear.

The lateral boundaries are, in the majority of cases, perfectly smooth, but columns and ridges running along the walls of the cyst have been described.

The ventricle is sometimes, though rarely, dilated as a whole; more frequently one horn only is the seat of disease and is more or less cut off by adhesions from the remainder of the cavity.

G. Vrolik<sup>53</sup> has described a specimen in which the anterior horn of one lateral ventricle was cut off as the result of a former inflammatory process. Dropsy taking place in the diverticulum thus isolated, the brain and corresponding part of the skull on the same side were greatly distended. Virchow<sup>54</sup> states that the posterior horn of one lateral ventricle may be obliterated either in its entire length or in part only, forming (encysted hydrocs of the posterior horn) a cavity which sometimes does not communicate at all with the ventricle and may then become the seat of morbid processes. The fourth ventricle is itself sometimes the seat of a limited effusion, and the same author has figured a localised and encysted dilatation of the fourth ventricle pressing on the facial nerve and producing a paralysis of the same. Axel Key and Retzius mention this case in their classical work, and suggest that this pathological condition was possibly due to the closure of the aperturæ laterales ventriculi quarti on the same side.

Hamel has seen unilateral vaso-motor paralysis of the face associated with congenital hydrops of the fourth ventricle. More lately Hemming,<sup>55</sup> writing in the *British Medical Journal* for 1887, reports a valuable case in which, at the *post-mortem*, he found the fourth ventricle distended by a cyst which occupied the whole cavity, had burrowed into the cerebellum and was filled with yellow serous fluid.

Hydrops of septum lucidum and pituitary body, with or without other effusions in the brain, have also been noted.

All these appearances must be, of course, distinguished from the cysts formed at the seats of an old hæmorrhage, &c.

In many cases of localised effusions in the horns of one of the ventricles, the dilatation gives rise to a hydro-encephalocoele. Some authors go so far as to say that the liquid in a hydro-encephalocoele is always due to dropsy taking place in a limited part of the cerebral ventricles. It is not intended to give here a full account of this variety of disease, but merely to show the relation existing between it and hydrocephalus.

In the vast majority of patients, the protrusion takes place in the occipital region, generally above the occiput, more rarely below it, or on the sides. In no less than 135 out of the 220 cases collected in Lawrence's,<sup>56</sup> Springs, and Larger's<sup>57</sup> papers, the hernia protruded in the occipital region. An important point is that the tumour does not pass through a fontanelle, as is sometimes described, but always through a bone. The frontal region, at the junction of the frontal and nasal bones, is another favourite place for its appearance; occasionally the protrusion takes place through the frontal bone itself.

Houel<sup>58</sup> states that the hernia may pass out through the following apertures: the os unguis at the level of the inner angle of the orbit, the sphenoid fissure, the body of the sphenoid or inner portion of its wings, the cribriform plate of the ethmoid and the sphenoidal sinus. Stein<sup>59</sup> has seen the tumour pass through the sphenoid fissure, follow the cavernous sinus on the right side, pass through the nose, and end in the mouth.

The centre of the tumour consists of part of the nervous

centres, the protruding part of course varying in size, the whole being covered by the red, thickened and villous pia mater. If the opening be small there is a distinct neck to the tumour; if large the cerebral hernia takes place *en masse*. The liquid accumulated in the ventricles pushes the cerebral matter before it, the convolutions gradually unfold, become thinner, adhere to the dura-mater and may even disappear. If the cerebral matter gives way, the herniated part collapses, forms a shrivelled stump, whilst the liquid is contained in a cyst on the exterior and may, through a fistula, communicate with the fluid inside. The ependyma, as a rule, shows signs of chronic inflammation. The same deformities of the body so frequently met with in cases of chronic hydrocephalus are often present in this variety. In only very few cases is there any mention made as to the state of the central canal of the cord, of the aqueduct of Sylvius, or as to evidences of obstruction in the central canal. In one patient the base of the brain is said to have been covered by a thick membrane, and in another there was a dilatation of the aqueduct of Sylvius, the foramen of Magendie being closed by a fibrous membrane.<sup>60</sup>

True chronic hydrocephalus differs from porocephalus only in the fact that the disease affects both ventricles without there being an actual loss of continuity in the cortical substance. The fluid when occupying both lateral ventricles often distends the latter to an enormous extent, these cavities occasionally containing as much as twenty-five pints of fluid. The ventricles lose their shape almost completely and, through destruction of intervening tissues, become converted into one huge cavity, the middle one being absorbed into the other two. The distension, however, is not uniform and is always more marked on one side or even in one horn of a ventricle than on the other side. In one brain examined by the writer, not only was the left side of the skull and brain distinctly larger than the right, but, on examining the left lateral ventricle, the middle part and anterior cornua were found to have been far more distended by the fluid than the posterior,

The corpus callosum is often displaced and forced upwards so as to lie on a level with the upper surface of the hemispheres. The nervous substance entering into its formation sometimes almost completely disappears and is then represented by a transparent<sup>61</sup> fibrous membrane only, which is thrown transversely across and presents in the middle line a kind of fold in which some fibres of the cerebral substance still exist. In one case this membrane was formed by a fibro-vascular layer continuous with the pia mater and a solid membrane lining the corresponding lateral ventricle.

The foramina of Monro, when both lateral ventricles are the seat of disease, are enlarged and measure sometimes as much as  $3\frac{1}{2}$  inches across. When the distension is unilateral the foramen on the same side has been found closed.<sup>62</sup>

The aqueduct of Sylvius is not wider in all cases—in fact its channel as well as the fourth ventricle are for the most part quite normal. The lining membrane of the ventricle shows well-marked pathological changes. It is often thickened and of a leathery consistence and either strips off too easily<sup>63</sup> or else is unduly adherent. It is rough, covered with small bands or columns running from side to side or with fibrous tracts and false membranes extending across the ventricles. These fibrous bands running along the walls of the ventricles are not unlike the columnæ carneæ of the heart. The surface of the membrane is shaggy, covered with small granulations or with small sago-like transparent bodies, the nature of which remains uncertain.

The most important point in the pathological anatomy of the disease, however, is the obstruction to the passage of fluid from one ventricle to the other, of which this thickened membrane may be the cause.

In a typical case described by Archambault,<sup>64</sup> a thick and resistant membrane formed the internal part of the walls of the ventricular cavities, numerous vessels and the cerebral substratum being visible below. This was simply the membrane normally covering the ventricles, but, being thickened, it was not unlike that found in the interior of a cyst. It lined the ventricular cavities, penetrated into the middle cavity through Monro's foramen and some pathological apertures,



passed over the orifice of the aqueduct of Sylvius into the third ventricle without dipping into it and thus closed the cavity.

This case illustrates the lesion most frequently found in hydrocephalus, namely, obstruction in some point of the canal running through the middle of the cerebro-spinal axis.

The obstruction may be lower down in cases where at the *post-mortem* the aqueduct and fourth ventricle are distended. In a specimen shown by Dr. Baxter<sup>65</sup> to the Pathological Society the cerebro-spinal opening was distinctly obliterated; for, on passing a probe backwards through the dilated iter a tertio, its point was felt to be stopped just below the calamus scriptorius by a thin but opaque membrane, the result of some former attack of very circumscribed meningitis. Magendie<sup>66</sup> himself has collected eight or ten observations of chronic hydrocephalus in which he had found the foramen first described by him closed by inflammatory adhesions, and Hilton,<sup>67</sup> in his lectures on rest and pain, mentions the same lesion as a cause of hydrocephalus.

Cruveilhier<sup>68</sup> states that Magendie's foramen was closed by a fibrous membrane in one case, and the same condition must have existed in Luton's<sup>69</sup> specimen. An obstruction, however, is not always present. It was distinctly absent, for instance, in the cases recorded by Mr. Hutchinson<sup>70</sup> and Gaucher.<sup>71</sup> In the specimen described by Mr. Hutchinson for instance, the cavity of a spina bifida was in direct communication with the brain by means of the enlarged central canal.

The choroid plexuses are often hypertrophied and their vessels diseased. In one patient the plexus choroideus medius running from the foramen of Monro to the posterior cornu was very thin, but the plexus here became as thick as a walnut, diminishing in size further on, the vessel walls being healthy. The plexus are occasionally bound to the adjoining tissues by inflammatory adhesions so strongly that their removal becomes impossible without inflicting injury on the neighbouring structures.

Cystic alterations in the connective tissue cells of the vessels' walls have also been described, and meningeal and other hæmorrhages in the cranial cavity are of frequent occurrence. The vessels likewise are often enlarged.



In a very interesting *post-mortem* examination of a hydrocephalic patient made by Dr. Dickinson,<sup>72</sup> an extra ventricle was formed by the tentorium and the upper surface of the cerebellum. This cavity was in free communication with each lateral ventricle by a large rounded opening on the outer surface of the transverse fissure on either side. The fourth ventricle and the passage leading to the third were perfectly normal, though there was some thickening of the arachnoid membrane between the cerebellum and spine, as also about the base of the brain.

The distension of the ventricles of the brain of a patient suffering from chronic hydrocephalus (age 50) which the writer had an opportunity of examining was more marked in the middle and in the anterior regions; consequently, the pathological changes in the convolutions were more evident anteriorly also. The cerebral substance over the frontal and orbital lobes was not more than one centimetre thick, whilst behind the Rolandic fissure the amount of brain-tissue gradually increased, so that, posteriorly, it amounted to double that in front.

The paracentral lobules were flattened, while the corpora striata and optic thalami appeared to be somewhat compressed, though otherwise normal. On examining the exterior of the brain the convolutions were still distinct; the sulci, however, much shallower than those of a normal brain. All the fissures were visible still, the sulci and convolutions in the occipital region being almost normal in appearance.

Distinct flattening of the ascending parietal and all the frontal convolutions, as well as of the paracentral lobule and the anterior part of the callosal and calloso-marginal convolutions, was noticeable; whilst the orbital part of the brain was almost normal in appearance. The island of Reil, however, had been forced outwards by the cerebro-spinal fluid and its surface lay level with the neighbouring convolutions, the whole of the insula being visible without any dissection. An obstruction to the flow of cerebro-spinal fluid could not be found anywhere.

The microscopic examination of this brain was made after hardening the organ in a weak solution of chromic acid, the pons, cerebellum and each hemisphere being treated separately. After hardening, small pieces were removed from the ascending first, second and third frontal convolutions, island of Reil, orbital, occipi-

tal, parietal and temporo-sphenoidal convolutions, also out of the angular gyrus, paracentral lobule, and sections made of these parts. The pons medulla, internal capsule and corpora striata were examined separately. The sections were stained in an ammoniacal solution of carmine, this fluid having been found to give the best results.

No lesions were found in the internal capsule or corpora striata and no tracts of degeneration in the pons or medulla. The only change found in the pons was that the central canal was almost, though not completely, filled with epithelial *débris* and that its margins presented an irregularly festooned border. The grey matter around it was softer than natural, breaking down easily and staining somewhat more deeply than the grey matter elsewhere, but no distinct histological changes could be discovered.

The cortical substance of the brain, however, showed well-marked pathological appearances, varying in character according to the different regions of the brain.

On holding to the light a section of the middle part of the ascending frontal, the grey matter, as is usually the case, appears to be stained more deeply than the white substance. Further, comparing this section with one from a normal brain, the layer of grey substance in the former is distinctly thinner by about one-sixth. On more careful examination, the grey matter is divided into three strata:—

1. A very small superficial stratum corresponding to the first cortical layer, stained somewhat less than the other parts of the grey matter.

2. A very thin stratum immediately underneath the first. This part contrasts sharply both with the stratum above and below, in staining far more deeply with carmine than the parts surrounding it.

3. The remainder of the grey matter which stains more deeply than the first, but far less than the second stratum.

On microscopic examination the first stratum consists of the first superficial layer of grey matter. This is perfectly healthy and normal in size and appearance. The second or deeply stained stratum is limited to the second layer of grey matter and contrasts very sharply with the parts above and below it. The deep colouration is due to a great excess of neuroglia, and when examined with a high power this layer is found to consist almost wholly of a very dense reticulum of fine connective tissue con-

taining a great many nuclei. The nerve-cells have almost completely disappeared in that layer, but the few which remain, judging from their size and shape, appear to be healthy.

The axis-cylinders have completely disappeared in some places. In other parts they are far less numerous than in a normal brain. This lesion is strictly limited to the second layer, the grey matter below, if somewhat reduced in amount, being otherwise quite healthy. The white matter is somewhat more vascular than usual, but no morbid changes of any kind are to be detected. The same lesion is also noticeable in the parietal convolutions, in the angular gyrus, in the island of Reil and in the paracentral lobule, as also in the callosal, calloso-marginal and marginal convolutions. In the island of Reil and paracentral lobule, however, the sclerosis though still limited to this second layer, is far more marked, the nerve-cells and axis-cylinders in the second stratum having completely disappeared in the grey matter covering the island of Reil.

In the occipital region the lesions are somewhat different. The first or superficial layer stains far more deeply with carmine than the regions previously described and microscopic examination reveals a considerable excess of connective tissue in this part. The second layer is quite healthy, but the third or intermediate stratum like the first shows a well-marked hyperplasia of connective tissue. The hyperplasia is not limited to this stratum, for in some places small microscopical bands of hyperplastic connective tissue extend from this layer into the deeper parts of the grey matter, where also the connective tissue is in excess.

The white matter below contains a considerable number of blood-vessels and shows an increase in the connective tissue. The nerve-cells are somewhat fewer in number in the places where the connective tissue is in excess, but the nervous elements do not seem to have suffered much, the axis-cylinders having a normal appearance and the nerve-cells, if somewhat less numerous, quite healthy.

No changes whatsoever can be detected in the sphenoparietal lobes, but in the orbital lobes distinct pathological alterations are found. Scattered through the first layer are a number of nucleated star-shaped cells, which are not nerve-cells, but connective-tissue elements forming little islands of sclerosis lying in the middle of healthy tissue. The nerve-cells in the deeper layers are surrounded by a large clear space and the cells and nuclei are filled with very fine granules—a lesion not unlike that which has been described as occurring in senile dementia. In the deeper parts these appearances are more marked still, the cells being

very granular and the spaces around them larger. But, strangely enough, there is no excess of connective tissue in this part of the brain. The optic nerves were examined, but no changes could be detected. To resume: bands of sclerosis were found extending over various parts of the brain. These bands affected one or several layers, according to the part of the brain affected, not spreading, as a rule, to the other layers. The nerve cells were not markedly affected except in one part, where they showed the lesions which are characteristic of senile dementia.

The interest of these lesions consists in the fact that a morbid process affecting the whole of the brain produces pathological changes varying in extent according to the part of the brain examined. Thus in the motor region the lesion is limited to the second, in the occipital region to three other layers. These changes resemble somewhat those found in the spinal cord where degenerations are often limited to certain definite tracts (motor sensory). It will be interesting to see, therefore, whether or not there is some correlation in the development or functions of some of these layers. Lastly, the lesions demonstrate the changes produced by long-continued pressure acting on the grey substance of the hemispheres.

The convolutions are often far more flattened than in the brain just described, the sulci becoming completely obliterated even, the whole brain being converted into one large membranous bag a few millimetres thick. Part of the brain or the membranes covering it may be forced out through a normal (fontanelle?) or pathological opening—through a bone even—and give rise to a true *hernia cerebri*.

The base of the brain, as a rule, is far less affected than the vault, or may be quite normal. The corpora striata, optic thalami, and other ganglia at the base are flattened and lengthened, or one corpus striatum and optic thalamus presents a normal appearance, whilst the other may have almost disappeared—another proof of the often asymmetrical distension of the head.

When the aqueduct of Sylvius is enlarged, the pons and cerebral peduncles are also flattened and altered in shape.

Virchow<sup>73</sup> has drawn attention to the presence of nodo-

sities of cerebral matter in cases of congenital hydrocephalus, these nodosities forming a kind of excrescence on the surface of the cerebral ventricles. They are especially met with in places where, in healthy brains, the surface of the ventricle is quite smooth—namely, just at the margins of the corpora striata and optic thalami. This condition, according to the same author, may be partly due to irritation. The membranes covering the brain are often somewhat thickened and occasionally adherent, presenting signs of recent meningitis,<sup>74</sup> but, as a rule, the lesions are by no means characteristic. Small pedunculated cysts hanging into the ventricles are occasionally present but of little importance.<sup>75</sup>

The cranium always presents characteristic lesions. In congenital porocephalus the head is normal in size, or even smaller than normal. Cruveilhier<sup>76</sup> has shown that premature ossification often takes place, the bones being frequently very thick and the sutures uniting too early. In chronic hydrocephalus the head may vary from just above normal to an immense size. The largest on record is a foetal head which was in Meckel's<sup>77</sup> possession, the biparietal diameter measuring forty-three centimetres, and another described by Frank, which measured 140 centimetres in circumference. These enormous dimensions are very rare, however. Vinsoneau<sup>78</sup> who has minutely examined this question, finds that the average hydrocephalic head exceeds the normal (1845 ccs.) by 244 ccs., and it is useless to enter into further details as to size.

A hydrocephalic cranium is always asymmetrical and measurement shows that one lateral half of the head is always larger than the other. The writer has accurately measured fourteen dried crania of hydrocephalic children and adults, and has found that measurement from the occipital protuberance to the root of the nose is always larger by three-quarter centimetres at least on one side than on the other, the difference on one occasion amounting to two and a-half centimetres, but averaging one and a-quarter centimetres.

Not only may one side be the larger, but the anterior part

may be increased out of all proportion to the posterior half, or *vice versâ*. Moreover, one very limited part of it, one or more bones, one frontal or occipital for instance, is sometimes expanded out of all proportion.<sup>79</sup>

The lateral distension is, as a rule, more marked than the antero-posterior. Thus Vinsoneau<sup>80</sup> found that out of thirty-two heads examined by him eighteen were brachy-cephalic, ten subbrachy-cephalic, and four dolicho-cephalic. The hydrocephalic crania may be divided into the following classes :—

Class I.—General enlargement.

Class II.—Partial enlargement.

(a) Anterior hydrocephalus, where the frontals are pushed forwards and irregularly ossified. (*Tête à bonnet à poils*. Giralès).

(b) Posterior hydrocephalus.

(c) Cases where the parietal bones being prematurely ossified, and the antero-posterior diameter being lengthened, the lateral parts chiefly give way (scapho-cephalus). Out of thirty-seven observations in Vinsoneau's paper only two showed this character.

(d) Cases where, owing to the partial enlargement of one ventricle, a few bones or only one are deformed.

The bones forming the vault of the skull are thin, as a rule, and transmit light readily, but in certain places they are sometimes thicker than natural. If the convolutions were still well marked at the time of death, their impressions, even to their minutest sulci, are sometimes marked on the bony skull. This was typically the case in one cranium which the writer, thanks to Professor Pierret's kindness, had an opportunity of examining at Bron Asylum.

On holding up the skulls to the light, places where the ossification is deficient are frequently seen. These points of deficient ossification, scattered more especially on the frontal and temporal bones are often merely covered by a lamina of bone, if not actually perforated. Large holes are often present in some of the bones, chiefly the frontals, through which a hernia cerebri sometimes protrudes.

The bones of the adult skull are often quite as thick,

or even much thicker than natural, though points where the ossification is deficient are often noticeable. The anterior fontanelle is sometimes covered by a thin layer of bone only, or the inter-frontal and other sutures are visible long after the time at which they usually disappear, showing that, in spite of the often enormous thickness of the skull, some further distension was still taking place at the time of death. The frontal, parietal, squamous portion of the temporal and occipital bones are those which suffer most. The frontal bones pushed forwards are often almost twice as large as natural, retarded in their union, or one or both of them are cleft through the middle. The enlarged parietals often differ in size and present the lacunæ above mentioned. In one cranium which the writer had an opportunity of examining the left parietal bone had evidently been divided into two, and traces of an accessory suture running from before backwards still remained at the time of death, which occurred at the age of fifty-four.

The occipital bone is almost always distinctly asymmetrical and, in children, a fissure often runs from its point of junction with the parietal bones to the occipital foramen. It is occasionally driven downwards and backwards so as to become almost horizontal. The foramen magnum is altered in shape, asymmetrical and oblique, on account of frequent malformation of the occipital bone.

Ossification of the sutures is occasionally premature, at other times retarded, both conditions existing in the same skull sometimes. Thus the writer has seen a dried skull of a child aged two and a-half years, in which the anterior fontanelle being still open and the lambdoid and coronal sutures still half a centimetre apart, the inter-frontal suture, which as a rule is the last to close in hydrocephalic children, was firmly ossified.

The sutures are sometimes linear, their margins being thin, almost straight and not dentated, a small fibrous band only showing between the bones. Occasionally they measure several millimetres or even centimetres in width. A chain of Wormian bones often forms in a suture<sup>81</sup> and keeps the bones apart. In other specimens the sutures appear to



be intimately joined, but nevertheless the bones can be disarticulated with the greatest ease.

Atypical sutures have also been described. The anterior fontanelle<sup>82</sup> often remains patent for a long time and its situation is often visible, even in the skulls of patients who died at an adult age. Cazalis mentions the case of a child 9½ months old whose anterior fontanelle measured 4½ centimetres across. In a girl sixteen years old Fürst<sup>83</sup> found the anterior fontanelle patent. The fontanelle of Gerdy and other accessory fontanelles are sometimes present, though the posterior and lateral fontanelles are generally ossified early.

Wormian bones are small accessory bones often present in the crania of hydrocephalic patients. They are small, generally placed on the sutures, often symmetrical and more frequently found on the posterior surface of the skull than on the anterior.<sup>84</sup> The lambdoid suture is their favourite situation, though according to some, they are most often present on the coronal and sagittal sutures. The face is almost always free of them. Vinsoneau,<sup>85</sup> however, describes one skull in which a Wormian bone was placed on the nasofrontal suture. There may be as many as ninety on one skull, but sometimes only one or two; their size, according to Sappey,<sup>86</sup> varying in an indirect ratio to their number. One Wormian bone placed exactly over the posterior fontanelle and articulating with the occipital and parietal bones, is probably only the highest part of the occipital which has remained distinct. It is a small lozenge-shaped or triangular bone often divided by fissures into two or three distinct portions (os epactale<sup>87</sup>).

Sir Prescott Hewett<sup>88</sup> describes a cranium in which the fore parts of the squamous portions of the temporals, as well as the great wings of the sphenoids, driven downwards and outwards, bulge into the zygomatic fossæ and lie on a level with the margins of the superior maxillary bones. The glenoid cavities in another skull are six inches, the two external angular processes of the frontal bones 7¼ in. apart. In one skull there is on each side a large pouch bulging outwards and projecting forwards into the region of the cheek,



as if the child's cheeks were blown out. The meatus auditorius externus is displaced to the under surface of the skull, below the level of the foramen magnum, and almost in contact with the condyloid process of the occipital. The anterior and middle fossæ of the skull are larger than natural, and defects in the ossification of the base of the skull, in the shape of premature or deficient ossification, are noticeable points in many skulls.

The orbital plates, especially in young children, are driven downwards and either present a plane surface oblique from before backwards, or are perpendicular, even convex, bulging into the orbit and sometimes reducing the latter to a mere chink. The orbital arch is more or less obliterated, the frontal and orbital portions of the bone sometimes forming one continuous line convex in its whole length. The orbits of adults, owing probably to the lesser degree of distension, are sometimes almost normal.

Though the face in these cases looks small on account of the enormous distension of the skull, it is quite as large if not larger than usual. The writer has measured the distances between the two malar bones and from the root of the nose to the chin in fourteen hydrocephalic skulls, and has found them to be quite as large as in normal children. In fact the distance between the two malar bones is as a rule increased by about 0c.5 in the hydrocephalic children measured by the writer. The face loses its oval shape and assumes that of a triangle with its apex at the chin. The root of the nose is often somewhat broad and the lower jaw prominent. In all cases examined by the writer the palate was somewhat narrow and high arched, the teeth were irregular, grooved longitudinally and often carious. The development of the teeth is not only retarded but premature caries is the rule. Bouchut<sup>89</sup> states that he has seen well-formed teeth in hydrocephalic children. This may be the case occasionally, but almost all the writers on the subject speak of the teeth as being retarded in their development and irregular in shape. In the Musée Dupuytrin in Paris there are more than twenty hydrocephalic crania. In many of them the teeth are completely missing, the alveoli having also disappeared, whilst in

the few in which the teeth are present they are invariably carious.

Many authors have noticed the coincidence of hydrocephalus with spina bifida, but the writer is unable to give any statistics as to the frequency of the coincidence. It has been reported that after the cure of spina bifida the child had become hydrocephalic, and it has been argued that the hydrocephalus in these cases was due to the cure of the spina bifida. It might, however, be argued with far more probability that the cases in which the symptoms were first noticed after the cure of spina bifida were cases in which the symptoms of hydrocephalus began some considerable time after birth. Dr. Barnes's<sup>90</sup> case is very interesting from that point of view, for though the spina bifida was congenital the hydrocephalus did not make its appearance till the patient was nine months old.

*Analyses of Cerebro-spinal Fluid in Cases of Hydrocephalus.*

TIDY. <sup>91</sup>				YVON. <sup>92</sup>			
Water	...	...	1000 parts	Sp. gr....	...	...	1008
Solids	...	...	5.18 parts				
				Organic matter...	...	...	6.100
				Mineral matters	...	...	9.1
				Fixed substances	...	...	15.1
				Albumen	...	...	.900
Albumen	...	...	.98	Total albumen	...	...	2.040
Sugar	...	...	.63	Urea	...	...	2.800
Carbonate of soda	...	...	.02	Fatty matters	...	...	.129
Sulphate of soda	...	...	.08	Phosphoric acid	...	...	1.291
Chloride of sodium	...	...	2.14	Sulphuric acid	...	...	.117
Chloride of potassium...	...	...	.6	Calcium	...	...	.089
Fat	...	...	.1	Chloride of sodium	...	...	7.
Alcohol extract...	...	...	.63	Water	...	...	994.900
			Total 5.18				Total 1000

HILGER. <sup>93</sup>				PAPP. <sup>94</sup>			
Water...	...	...	98.775	Sp. gr.	...	...	1007.078
Solids...	...	...	1.225				
Salts ...	...	...	.762	Chloride of sodium	...	...	.32%
Albumen ...	...	...	.246%	Carbonate of soda	...	...	.41%
Sugar ...	...	...	.164%	Phosphate of soda	...	...	.02%
				Phosphate of calcium...	...	...	.01%
Chloride of sodium	...	...	.397				
Chloride of potassium	...	...	.682				
Sulphate of potassium	...	...	.032				
Phosphate of potash	...	...	.124	or			
Phosphate calcium	{	...	.096	Soluble salts	...	...	.706
Magnesium				Alcoholic extractives	...	...	.004
Iron				Albumen	...	...	.181
				Water	...	...	99.049
Total			100	Total			100

Other deformities sometimes associated with hydrocephalus are hare-lip, cleft palate, absence of thoracic and abdominal organs, transposition of organs, talipes, pes equinus, equino-valgus or varus, double fingers, malformation of arms, legs and feet, or absence of one or more bones.

## LIST OF REFERENCES.

- <sup>1</sup> Littré, *Œuvres complètes d'Hippocrate*, Vol. vi. et vii.
- <sup>2</sup> *Ibid.*, Vol. vi.
- <sup>3</sup> *Ibid.*, Vol. vii., p. 127.
- <sup>4</sup> Galen, Book xiv., p. 782. *Lipsii in off. libr. Caroli Cnoblochii.*
- <sup>5</sup> Celsus. See Transl. by Ninnin, Paris, MDCCCLII., Vol. i., p. 323.
- <sup>6</sup> Oribasius, Darenberg's edition, Vol. i.
- <sup>7</sup> Hildanus de Hilden, quoted by Itard, *Diet. des Sc. Med.* Article "*Hydrocephale.*"
- <sup>8</sup> Vesalius. See Morgagni, *De sedibus et causis morborum.*
- <sup>9</sup> Fabricius. See Ambroise Paré.
- <sup>10</sup> Ambroise Paré, *Œuvres complètes*, p. 186, Vol. i.
- <sup>11</sup> Morgagni, *De sedibus et causis morborum*, *Epist. anat. med.* XII.
- <sup>12</sup> Wepfer, *Observations Medico. pract. Schaphusii*, 1727.
- <sup>13</sup> De la Motte, *Traité des accouchements*, Paris, 1721.
- <sup>14</sup> Smellie. See Poulet.
- <sup>15</sup> Solomon Naumann, *De partu. diffic.*, Leips., 1762.
- <sup>16</sup> Murray, *Thesis*, 1797.
- <sup>17</sup> Baudelocque, *Art. des Accouchements*. See also Poulet.
- <sup>18</sup> Simpson, 'Obstet. Works,' London, 1856
- <sup>19</sup> Cazeaux, *Bull. Soc. de Chir.*, Paris, 1855-56.
- <sup>20</sup> Tarrier, *Thèse*, Paris, 1860.
- <sup>21</sup> Depaul, *Med. Times and Gazette*, 1874.
- <sup>22</sup> Chassinat, *Gaz. Med.*, 1864.
- <sup>23</sup> Ramsbotham. See Poulet.
- <sup>24</sup> McDonald, *Ed. Med. Jour.*, 1878, and *Trans. Ed. Obst. Soc.*, 1878.
- <sup>25</sup> Fritsch, Halle, 1876.
- <sup>26</sup> Klebs, Praag, 1876.
- <sup>27</sup> Poulet, *Thèse de Paris*, 1880.
- <sup>28</sup> Tulpus, orig. could not be discovered, quoted by several authors. See Morgagni.
- <sup>29</sup> Wepfer, *Loc. cit.*
- <sup>30</sup> Bonnet, *Loc. cit.*
- <sup>31</sup> Valsalva, orig. could not be obtained. See Morgagni.

- <sup>32</sup> *Loc. cit.*  
<sup>33</sup> Lieutaud. See Itard, *Loc. cit.*  
<sup>34</sup> Whytt, 'Obs. on Dropsy of the Brain,' Ed., 1768.  
<sup>35</sup> Warren, quoted by Itard, *Loc. cit.*  
<sup>36</sup> See Itard, *Diet. des Sc. Medic.*  
<sup>37</sup> Hilton, 'Lect. on Rest and Pain.'  
<sup>38</sup> Kundrat, *Die Porencephalie*, Gratz, 1882.  
<sup>39</sup> See 'Quain's Anatomy' and 'Kolliker's Embryology.'  
<sup>40</sup> Cruveilhier, *Anat. Path. du corps humain*, L. 15, Pl. 4, and L. 39, Pl. 4.  
<sup>41</sup> Morgagni, *Loc. cit.*  
<sup>42</sup> Cruveilhier, *Loc. cit.*  
<sup>43</sup> Schwarz, *Bull. Soc. Anat.*, 1873.  
<sup>44</sup> Budin, *Soc. Anat. de Paris*, 1875.  
<sup>45</sup> Duret, *Bull. Soc. Anat.*, 1875.  
<sup>46</sup> Dickson, *Lancet*, 1838.  
<sup>47</sup> Binswanger, *Berlin Klin. Woch.*, 1883. *Virch. Arch.*, Vol. cii.  
<sup>48</sup> Frigario, *Ann. Univ. di Med.*, 1887.  
<sup>49</sup> Cunningham, *Journal of Anatomy*, Vol. xiii., p. 508.  
<sup>50</sup> Schwartz, *Loc. cit.*  
<sup>51</sup> Kundrat, *Loc. cit.*  
<sup>52</sup> Cotard, *Thèse de Paris*, 1868.  
<sup>53</sup> G. Vrolik, *Traité sur l'hydrocéphalie interne*, Amsterdam, 1839.  
<sup>54</sup> Virchow, 'Path. of Tumours,' Vol. i.  
<sup>55</sup> Heming, *Brit. Med. Jour.*, 1887.  
<sup>56</sup> Lawrence, *Med. Times and Gazette*, 1858.  
<sup>57</sup> Spring. See Houel.  
<sup>58</sup> Houel, *Soc. de Biol.*, 1863.  
<sup>59</sup> Stein, *Ibid.*  
<sup>60</sup> See Cruveilhier, *Loc. cit.*  
<sup>61</sup> Archambault, *Bull. Soc. Anat.*, 1852, 1856, 1863.  
<sup>62</sup> This statement appears in several text-books, but cases in which this condition was described could not be discovered.  
<sup>63</sup> Archambault, *Loc. cit.*  
<sup>64</sup> Baxter, *Trans. Path. Soc.*, London, 1880-81.  
<sup>65</sup> Archambault, *Loc. cit.*, 1854.  
<sup>66</sup> Magendie. See Axel Key and Retzius.  
<sup>67</sup> Hilton, Lectures on 'Rest and Pain.'  
<sup>68</sup> Cruveilhier, *Loc. cit.*  
<sup>69</sup> Luton, *Comp. R. Soc. Biol.*, 1857.  
<sup>70</sup> Hutchinson, *Trans. Path. Soc.*, 1859.  
<sup>71</sup> Gaucher, *Soc. Anat.*, 1879.  
<sup>72</sup> Dickinson, Lectures on 'Hydrocephalus,' *Lancet*, 1870.  
<sup>73</sup> Virchow, 'Path. of Tumours.'  
<sup>74</sup> See Dickinson, Baxter, and many others.  
<sup>75</sup> Wenor, *Lancet*, 1855, and others.  
<sup>76</sup> Cruveilhier, *Loc. cit.*  
<sup>77</sup> See Pouillet, *Thèse d'agrégation*, 1880, Paris  
<sup>78</sup> Vinsonneau, *Thèse de Paris*, 1880.  
<sup>79</sup> G. Vrolik, *Loc. cit.*  
<sup>80</sup> Vinsonneau, *Loc. cit.*  
<sup>81</sup> Gripat, *Soc. Anat.*, 1873.  
<sup>82</sup> Cazalis, *Soc. Anat.*, 1881.  
<sup>83</sup> Fürst Virchow's *Archiv.*, 1884.  
<sup>84</sup> Vinsonneau, *Loc. cit.*  
<sup>85</sup> *Ibidem.*  
<sup>86</sup> *Ibidem.*  
<sup>87</sup> *Ibidem.*  
<sup>88</sup> Prescott Hewett, *St. George's Hosp. Rep.*, Vol. i.  
<sup>89</sup> Bouchut, *Lec. clin.*, Vol. i.  
<sup>90</sup> Barnes, *Trans. Obst. Soc.*, London, 1863-64.  
<sup>91</sup> *Lancet*, 1869.  
<sup>92</sup> See Bourneville.  
<sup>93</sup> *Centralblatt*, 1867.  
<sup>94</sup> *Jahrbuch f. Kinderheilkunde*, 1867.

# BRAIN.

PART II., 1890

Original Articles.

## THE PSYCHO-PHYSICAL PROCESS IN ATTENTION.<sup>1</sup>

BY J. SULLY.

*Read before the Neurological Society on December 19, 1889.*

I PROPOSE to discuss a subject that seems at first sight of more consequence for the pure psychologist than for the neurologist. I hope, however, that I may succeed in showing that the topic has its importance for all who make a study of psychical phenomena: whether, as psychologists, they are primarily interested in these phenomena on their own account, or, as neurologists, they deal with them rather as concomitants and symptoms of those nervous processes with which they are more immediately concerned.

I may probably assume here that we are agreed upon the possibility of a true science of psychology, or, if the expression is preferred, physiological psychology, which proceeds by bringing together as closely as possible the results of subjective and objective research. And here I trust I may not seem presuming if I remark that in this work of co-ordinating the two kinds of knowledge—that of our conscious states and that of the functional activities of the nervous system—there must be on either side a certain give and take. On the one side the psychologist should welcome every new discovery that helps to elucidate the obscure and subtle actions of nerve and nervous centres. On the other side, he may rightly claim that a certain respect be paid by the neurologist to the results of psychological observation and

<sup>1</sup> It is hoped that a discussion of the subject of the paper will appear in the next number.

analysis. We must remember that in this conjoint investigation of psycho-physical processes from the subjective and the objective side, the psychologist has had the advantage of a start. With respect to many of the more complex phenomena involved, every candid neurologist will, I believe, admit that the knowledge of the physiological factor is meagre and mostly conjectural. And it is precisely here that the psychologist claims to have done something in the way of simplifying by analysis the tangle of complexity. Not that the candid psychologist—for I trust that there are such to be found—would contend that his results are above suspicion. Every fair-minded student of psychology will at once grant that the history of the so-called science presents much more a congeries of individual systems than one organised body of verified knowledge. All that he will ask, if he is reasonable, is, that while he is gratefully accepting the fruits of neurological research and seeking to adjust them to known mental processes and laws, the neurologist should pay him the reciprocal courtesy of pursuing his researches in the light of the best and most assured results of psychological analysis.

These remarks seemed to me almost necessary in introducing to a gathering of neurologists the subject of Attention. Attention, in its higher form of voluntary selection and fixation of particular presentations of sense and ideas, is apt to be thought of as a mysterious emanation of spiritual activity that interferes with and controls the mechanism of mind. According to this view, it is the occult *Ego* that asserts itself in the activity of attention, and by scanning the given contents of the moment, and by separating and re-arranging these, is itself the author and creator of its higher intellectual life. Attention, in fact, has been involved in all the psychological mysticism which has clouded the processes of volition. The spiritualist in psychology who fights hard for the spontaneity and liberty of the subject against the upholders of mechanical uniformity, naturally betakes himself to the lofty region of voluntary attention as an inexpugnable akropolis. And even some writers on the physiology of mind have treated the whole process of

selective attention as standing outside the circuit of psychophysical processes—an alien presence, “mystic, wonderful.” As a natural reaction against this conception of attention as a recurring miracle, the physiologist has, I suspect, been disposed to ignore it, or, at least, to under-estimate the large part played by it in the mental life. He may not have gone so far as Condillac, who defined attention as nothing but the transformation which a sensation undergoes when it predominates by its vivacity. Yet he has, I think, tended to overlook its distinguishing characteristics and its common co-operation in our psychical processes. “Sensations (he has seemed to say) I know, and movements I know ; but who are you ?”

But a *rapprochement* of the psychologist and the neurologist is recognisable here also. The latter has come to recognise that our sensations, in their vividness and persistence, are determined not only by conditions in the peripheral process of stimulation, but by a reflex central reaction. This, indeed, naturally follows from the conception which seems to be now commonly adopted, that every psychophysical process is at once sensory and motor. A good deal has been done by Fechner, Bain, Wundt, Ferrier, and others towards determining the precise nature of this nervous reaction. The claims of attention, moreover, have not been lost sight of in assigning special cortical seats to the several distinguishable psychical claimants. In England, as well as in Germany, the question of the precise region of the cortex involved in the process of attention has been the subject of considerable discussion.

In view of this hopeful state of things, I venture this evening to urge the claims of the subject on neurologists. I need hardly add that I shall endeavour to conceive of attention as a process at once psychical and physical, of which we may, by properly scientific methods, determine the co-operant factors and conditions.

I propose to give at the outset a brief *résumé* of some of the more important results of the psychological treatment of the subject ; then to see what recent experiment has done to make these results more complete and exact ; after this, to

consider the current views respecting the nervous process involved; and, lastly, to point out some of the practical bearings of the subject, and more particularly its relation to healthy brain-action and to normal life.

(A) *Psychology of Attention.*

I must here confine myself to a few of the more important points.

In the first place, then, I think it may be said that recent psychological research has tended to the conclusion that the common and typical form of psychosis is complex in which a presentative element of sense, or its ideational equivalent, is followed and reinforced by a distinctively active element of attention—a third element of feeling, that is pleasure or pain, being commonly, if not in all cases, interposed between them. According to this view the simplest fact of consciousness, when it presents itself in a complete and distinct form, is determined in part by a mental reaction. In many cases the two factors—sensation and reaction—are easily distinguishable, as when attention follows slowly on the persistence of a sub-conscious sensation, as, for example, in listening to the repeated stroke of the clock or other prolonged sound, or in directing attention to some obscure organic sensation; or when, on the contrary, the direction of attention precedes the occurrence of the sensation as a process of mental preadjustment or expectant attention. In other cases it is less easy to separate the two constituents; for instance, when surprised by a loud sudden sound, or mastered by a teasing and persistent idea. Closer inspection will however show in these cases, too, that the completed result involves a reaction. A sudden sound is not distinctly heard for an appreciable interval, during which the reactive process presumably adjusts itself. And in the familiar experience of going back unwillingly at night to some worry of the day, it is easy to see that not only do the ideas recur, but that after a vain struggle to exclude them perhaps we proceed to react in the customary way, just as if it was of the first importance for us to settle the perplexing point at this very moment.



The psychologist distinguishes this reaction as having the characteristics of an *active* phase of consciousness. This he does solely on the ground of its qualitative peculiarities, and without any *arrière-pensée* of an active spiritual principle. Attention is an active phenomenon, in precisely the same sense as conscious movement is active. It presents in its several variations all the known active elements, as craving, effort, attainment. This active phase becomes most distinct in those more complex forms of the process commonly marked off as voluntary or volitional attention, where there is a keen sense of resistance and struggle—as when by a special effort a man attends to a matter when suffering great bodily discomfort, or plagued by some disturbing noise.

The consequence and evidence of the completed process of attention or active mental adjustment is the fixation of the particular sensation. Attention is Detention in consciousness.<sup>1</sup> This detention instantly brings about or is attended by a certain rise in vividness or intensity, and also by a very considerable increase in distinctness. When I attend, for example, under favourable circumstances to a sound, I make it for the time the predominant mental content. I may just add in passing that the rise in intensity and in definiteness together, due to attention, differs from the rise in intensity due to an external cause in two particulars: (a) The rise in intensity is narrowly limited; (b) it subserves and seems subordinated to the increase in definition. A musical sound of a given pitch does not go on becoming more definite in quality as it gets louder; but, on the contrary, after a certain moderate intensity, becomes less definite. But attention uniformly tends to definiteness of quality.

Lastly, psychology tells us that attention has some at least of its conditions among conscious phenomena. The simplest supposition would be of a sensation calling forth a reflex attention in the absence of all contending forces. This idea is of course never realised. At no moment is our

<sup>1</sup> This is clearly illustrated by Prof. Ribot in his recent volume, *La Psychologie de l'Attention*.

consciousness reduced to a single presentative element. But there may be an approximation to this state of things, as when surprised by a noise in the silence of the night. In such a case we say the reaction is the direct result of the sensation itself. Among the features of sensation which have most to do in determining this reflex attention may be named intensity, suddenness, or unexpectedness, also duration, including recurrence, as in the case of the clock-strokes. The effect of suddenness, as mental shock, is familiar. A sound, if gradually introduced, might fail to call forth attention, even when attaining a considerable intensity. It may be remarked further that all increase in intensity does not further the reactive process. A very loud sound "overpowers us," as we phrase it—that is, baffles the adjustive process of attention.

The first step in the complication of conditions is where the sensation is distinctly pleasurable or painful. Here attention is prompted by feeling, and begins to follow the directions of interest. I here assume that both pleasure and pain have at first the effect of rousing attention. This is, I think, borne out by observations of familiar facts, as children's and grown-up people's susceptibility to the fascination of the horrible, and the vast capacity of most people to make themselves supremely unhappy. There seem to be good reasons, too, from a biological point of view, why we should attend to painful sensations, which in most cases at least indicate a harmful action on the organism. The preferential attention to pleasurable sensations is, I conceive, an acquisition involving volitional selection.

The next stage of complication is the partial determination of the direction of attention by pre-existent lines of interest, involving the residua of previous experience, and habitual forms of mental activity. That as we grow older all the more vigorous sort of attention tends to confine itself within certain limits answering to our special pursuits, information, and tastes, is a trite observation. We each see what we are prepared, by the special circumstances of our life, to assimilate or understand, feel the worth of, or turn to practical account. This cumulative effect of the past on the

present in fixing predominant lines of interest has not, I think, been adequately dealt with by English psychologists. The Herbartians in Germany treat of this under the head of that much-abused word *Apperception*.

The last stage in the complication of the process, is what psychologists mark off as voluntary or consciously selective attention. Here a number of forces is supposed to be at work, and attention is prompted hither and thither. There emerges the complex psychical phenomenon, a conscious resolve to attend to one particular sensation or idea under the stress of a predominant desire or motive. This volitional process has for its distinguishing accompaniment what is commonly spoken of as mental effort, and sense of resistance.

#### (B) *Recent Psycho-physical Experiments.*

Our knowledge of the process of attention, its variations, and its effects, has been materially increased by recent psychological and psycho-physical experiment. Attention is a subject on which the psychologist may expect the physiologist and the scientific worker in general to throw some light. Observation, as we all know, implies attention in its highest and most disciplined form, as steady, prolonged, mental concentration on the phenomena to be recorded. Hence it is not surprising that valuable psychological facts relating to the working of attention have been contributed by physicists, more particularly, observers of optical phenomena. I may refer to the apparent changes in the appearance of optical phenomena due to variations of attention, as the transformation of relief in a drawing, according as the observer attends to the one or to the other aspect, and the quenching of one coloured field by another, when the observer fixes his attention on this last.

It is, however, in a long series of experimental inquiries into what is known as reaction-time that the effects of attention have been most fully elucidated. There is no need to attempt any detailed account of these here, as they have already formed the subject of a paper contributed to *BRAIN*.

I will therefore content myself with a reference to one or two of their main results.

One of the most valuable results brought out by these experiments, is, I think, the fact that attention to an impression of sense is a process of adjustment which occupies an appreciable and measureable time, this time being longer or shorter according as the conditions of adjustment are unfavourable or favourable. Thus to take an unfavourable case, it is found that the reaction-time is appreciably lengthened when a disturbing cause, *e.g.*, an organ playing in the same room is at work; in the case of a person whose normal reaction-time was  $100\sigma$  (where  $\sigma$  is  $\frac{1}{1000}$ th part of a second), it rose under these circumstances to  $148\sigma$ . If the subject again is taken completely off his guard it may go up to  $500\sigma$  (half a second). On the other hand, if the process of adjustment is carried out to some extent beforehand, the reaction-time is reduced. For instance if the subject knows by some signal the instant at about which the impression is to arrive, the reaction-time may fall from  $253$  to  $76\sigma$ . When the preadjustment is complete an illusory perception with respect to the exact moment of the occurrence of the impression may arise, and the subject react too soon.

These experiments have been elaborated and varied by Wundt and his pupils in different ways. Thus, instead of a simple, sensory stimulus, on which the same reaction—say a movement of the hand—uniformly follows, one of a number of stimuli is applied, and the subject required to carry out one of a number of movements, *e.g.*, of the different fingers corresponding, by previous arrangement, to the particular stimulus selected by the experimenter. In this way Wundt considers that he has been able to determine the duration of different psycho-physical factors in complex processes, such as discrimination time and volition time.

These experiments, interesting as they are, have not yielded such certain results as one could wish, for Wundt's own interpretation of the facts reached is not quite satisfactory, owing to the language he employs in describing the process of attention, or as he calls it, apperception. His

whole theory of apperception has been severely criticised by Dr. Bain, and more recently by Dr. Hugo Münsterberg in a very remarkable publication, *Beiträge zur Experimentellen Psychologie*. Münsterberg has worked out a series of experiments himself and claims to have demonstrated that complex process involving rudimentary acts of judgment can be carried out under certain conditions in a purely mechanical way, and without the intervention of Wundt's indeterminate element, the selection of a particular impression by a volitional act of attention, and of the corresponding movement by a volitional impulse. Dr. Münsterberg effects this by getting the subject of the experiment, after a careful preliminary association of the several kinds of sensory signals (here words), and the corresponding finger-movements, to divert the attention altogether from the expected sensory signal and to concentrate it on the movements. Under these circumstances he finds that the reaction-time does not increase with the complication of the process as Wundt contends, but remains approximately constant. In these cases, then, there seems, as Münsterberg argues, no room for the discriminative and the volitional periods. The whole process becomes indeed largely unconscious or sub-conscious.

I have referred to this latest outcome of psychological experiment in Germany because it seems to me to illustrate in a new and striking manner the effect of pre-adjustment. The possibility of the whole process lapsing, in this instance into a quasi-unconscious one, is plainly due to the circumstance that the work of attention has been done in advance. Thus the subject passes, say, from the sound of the Latin word *lup-o* to the movement of the particular finger agreed upon as the digit which is to respond to the dative case, because in the preliminary mental fixation of the dative case and the associating of this with that particular finger-movement, the sound of the inflexion *o* was included. Similarly in the case where the signal "Schiller!" is instantaneously followed by the particular movement which has been already co-ordinated by a severe preliminary effort of mental concentration with the sound of the name "poet."

(C) *The Nervous Process in Attention.*

We may now pass on to consider the exact nature of the physiological process in attention. That attention stands in a particularly close relation to the process of motor innervation is a fact that discloses itself to even superficial observation. If we take the simplest form of attention, that directed outward to an impression of the sense, it becomes obvious that the mental adjustment is effected, in part at least, by a muscular adjustment. Thus, in the case of expectant attention, when we are awaiting an impression of sight or of hearing, it is easy to recognise in the pose of the body, the movement of the head and eyes, the presence of a vigorous motor factor. This factor does not manifest itself exclusively in actual movement. One would rather say that the characteristic of this muscular exertion is fixation of body, limb, and sense-organ, by a balanced action of antagonist muscles, and by the inhibition generally of all diffused movement.

In the case of fixating ideas the motor factor is less patent than in that of sensuous attention; but it is nevertheless present and observable. Fechner was one of the first to give a careful account of the muscular sensations localised in the head which accompany the effort of thinking. Others, as G. H. Lewes, have layed stress on the part played by the respiratory muscles in the severer acts of intellectual concentration. Dr. Bain goes farther, and seeks to account for the whole process of thought-control by help of a motor process. According to him, every idea is compounded of an element of passive and of muscular sensation. When, by an effort of will, we fixate or transfix the idea, it is because we can by the proper mechanism of volition, the motor apparatus, act upon the muscular factor—that is, I presume, strengthen and intensify it by a complete or partial renewal of the original movement.

In considering the sufficiency of the hypothesis that attention is a motor phenomenon, pure and simple, two points need to be examined: (1) Does it harmonise with what we know subjectively respecting attention? (2) Does it account for the known effects of attention?

On the first of these points I have little to say. That there is a close affinity between muscular and mental exertion I readily grant. Each is a variety of the active phase of consciousness. This affinity is illustrated by the fact that psychologists of eminence as Wundt in Germany, and Ward in England, regard both modes of activity as at bottom one.

And here may I be allowed to say, in passing, that from a psychological point of view, the peculiar quality of the psychical experience which we mark off as active, strongly suggests that muscular sensations are not wholly correlatives of afferent nerve-processes. If every psychical difference of quality is to have its corresponding physiological difference, there is some *a priori* reason for correlating the great and profound difference between passive sensation and active consciousness with the most important and radical difference observable in the nervous process—viz., that between the afferent and the efferent current. As, however, the subject of muscular sensation has been discussed so recently by this Society I should be sorry to complicate this evening's topic by raising again this difficult issue.

I will pass, then, to the question, whether what we all know as the mental attitude of attention appears subjectively identical with muscular consciousness.

Now, I think that if we carefully examine our state of mind when we are attending to impressions or ideas, we are in some cases able to distinguish attention as something apart from and independent of muscular activity. Thus, as everybody knows, we may fix the eye on an object and yet not attend to that object. Helmholtz notices the fact that we can attend to an object in the side regions of the field of vision without fixating the object. Where, one may ask, is the motor factor, the process of muscular adjustment in this case?

It may no doubt be said that there is a *tendency* in this case to move the eye in the direction of the object. But this tendency may, I believe, be completely counteracted without any injury to the process of attention itself.

In this connection I should like to refer to a personal experience that befel me while I was thinking over this



subject. I was walking along a narrow lane in a fog, lost in thought. I came on a lamp which shot its rays through the fog. I involuntarily stood still and fixated the lamp, thinking all the while intently on my psychological problem. When this intellectual effort relaxed, and not till then, I saw and recognised the lamp. Now here was energetic muscular action, and equally energetic concentration; but what was the relation between the two? That this involuntary assumption of the attitude of the seer, of the fixed head, convergent eyes, &c., somehow aided the process of mental concentration is certain. But was this muscular adjustment the *whole* of the process? If so, I ought surely to have been mentally occupied with no abstruse problem of psychology, but with the concrete sensible object before my eyes, even though it had been far less brilliant and imposing an object than the lamp. It struck me that I had, by pure good fortune, lighted on a case where the two phenomena, though both present, were rendered exceptionally distinct. The whole pose of body, head, &c., was for seeing; yet I saw not because that which seems something else, and to which I give the separate name of attention, was elsewhere.

The same partial independence of the process of attention and of the motor process appears, too, in ideational attention. If I think, for example, of a circle, with the eyes closed, I am aware of what Dr. Bain calls a control of the muscular element. I not only have muscular sensations which tell me that the peripheral organ by means of which I acquired the idea is engaged, but I am aware of a motor impulse to retrace the curve of the circle. It has been observed by N. Lange that in recalling the image of an object there are commonly observable movements of the eye corresponding to the contour of the object.<sup>1</sup> But now let us suppose that I am trying to visualise not any particular form, but merely some shade of colour—say peacock-blue. In this case, I certainly find the motor element much less prominent. I am hardly aware, indeed, in this instance, of any sensation of ocular strain, and should rather say that the eyes were in the easy and natural possession, described by Helmholtz as

<sup>1</sup> Quoted by Wundt, *Physiol. Psychol.*, ii. p. 242.



the primary position. Nor does attention in this case resolve itself into a renewal of the muscular action concerned in uttering the name of the colour. Indeed, I find that any thought of the name in this case, so far from helping, distinctly disturbs the visualising process. I must for the moment be all internal vision. And yet, though the muscular element in this case is, to say the least of it, considerably reduced, the active consciousness, the attention, is as clearly present as before.

On purely subjective grounds, therefore, I should be disposed to say that attention, though always accompanied by a motor process, which last, through the concomitant muscular sensations, undoubtedly lends something of its characteristic colouring to the mental state, cannot wholly be resolved into a motor phenomenon. The relation of the two is, I suspect, very similar to that which obtains between an emotion and the several sensory and motor phenomena which accompany it. Fear is always accompanied by characteristic physiological changes, involving the organs of circulation, respiration, and the muscles; and fear would not be fear but for these processes which contribute, in the sensations to which they give rise, characteristic features to the mental state. Yet when Professor W. James, of Harvard College, not long since proposed to prove that emotion is nothing but the result of the organic changes and correlated sensations, most persons probably regarded the proposal as paradoxical.

(2) Is this conclusion, reached by subjective analysis, confirmed by a consideration of the known effects of attention? And here we may confine ourselves to the most immediate and palpable effect of attention—the intensification and definition of the sensation or idea.

In the case of actual sensations obtained through the special sense-organs, the motor process must of course be pronounced sufficient. That the muscular adjustment of the eye and head for direct and clear vision, of the ear and head for sounds of a particular direction and definite pitch should help to strengthen and define the sensation, is just what we should expect. Yet even here muscular adjust-

ment does not in all cases—for example, that of attending to an object in the side region of the field of vision—seem to be an adequate cause of the phenomenon.

Again, in the case of attending to organic sensations, it is easy to see that a motor process may be present, which would serve, to some extent at least, to produce the observed effect. If, as is generally allowed, we learn to localise our bodily sensations by help of movements, which bring the hand to bear directly or indirectly on the particular region of the organism, it would seem to follow that in fixing consciousness on any part, motor elements of a nascent character may concur.<sup>1</sup> Yet it hardly seems likely that such a faint motor constituent could effect the remarkable results here observable. And, indeed, as I understand, physiologists refer the phenomena mainly, if not exclusively, to another process, also motor, though not implicating the so-called voluntary muscles, by which the circulation of the part attended to is modified.

Let us now come to the more difficult case of ideas. The theory that a motor process effects the fixation and increase in vividness and clearness of the idea seems to imply that in attending to an idea there is a process of motor innervation corresponding to that which subserves the development of the correlative sensation, and involving therefore the particular peripheral organ concerned. Thus, in seeking to visualise a colour the ocular muscles are supposed to be innervated. The further explanation of the effect as given by Wundt and others is as follows. As the result of the innervation of the ocular muscles, muscular sensations arise, which, being associated with the original sensation of colour, serve to reinstate this in a much more vivid ideal form.

In justice to the view that would resolve the whole of attention into a motor process, I may point out that the effect of motor innervation on ideation may be wider than is here suggested. If, as is fairly certain, the ideational

<sup>1</sup> It is worth noting, perhaps, that the range of co-operation of motor elements is materially increased by the supposition that there is an innervational stage of active or motor consciousness, for this might arise by means of a weak motor discharge which does not involve actual muscular contraction.

centre stands in the closest organic connection with the corresponding sensational—if indeed the two are to be regarded as distinct—and this last again, in the closest organic connection with the motor centre for the sense-organ, it seems conceivable and probable that any excitation of this motor centre, even if it did not effect actual contraction of muscle and reflex muscular sensation, would react on and reinforce the activity of the ideational centre.<sup>1</sup>

It may, I think, be granted that we never think of a colour, sound, or smell without some amount of innervation of the muscles of the corresponding peripheral organ. Subjective reflection tells us that this is so. But as we have seen, the motor process involved seems, in certain cases, to be a very faint one. It is to be observed, too, that the muscular sensations most characteristic of ideational attention or thought are not those of the sense-organ concerned, but of the head. According to Fechner they are the concomitants of contractions of the muscles of the skin on the occipital region of the head.

It has recently been suggested by Münsterberg, in the work already alluded to, that these muscular sensations of the head are concomitants of that fixation of the head which is so important a factor in the muscular adjustment involved in looking or listening. But so far as I can observe, the actual operation of looking or listening is not accompanied by any corresponding contractions of the muscles of the skin of the head.

Let us now inquire more particularly into the effects of such muscular action in the peripheral organ as is known to take place in ideational attention. That the innervation of the ocular muscles does favour visualisation is I think proved by a number of facts, more particularly that steadily gazing at the fire, the distant horizon and so forth, tends to develop a state of vivid reverie, which in extreme cases may rise to the strength of a hallucination. But I think it is easy to see that the whole effect in these cases is not due to the muscular sensations.

<sup>1</sup> Since writing this I have noticed a passage in Dr. Ferrier's *Functions of the Brain* (second edition, p. 462), which seems to me to recognise this direct action of motor on sensory centre.

And here it is important to distinguish between those sensational and corresponding ideational elements which merely involve a *general* muscular adjustment of the sense-organ concerned, and those which in addition involve a *special* muscular action. Thus in the case of visual elements we have colours which simply presuppose adjustment of the eye for seeing under particular conditions, and forms which involve definite muscular movements of the organ corresponding to the lines composing the form. That in recalling the form of a circle, the renewal of the muscular action should tend to reinstate the idea is just what might be expected. It may be remembered here that J. S. Mill made use of this circumstance in explaining our intuitive certainty with respect to the truth of geometrical axioms. We can at any moment, by ocular movement, re-create the experiences out of which our first ideas of straight lines, circles, and so forth were extracted.

In the other case, represented by the visualisation of colour, the adequacy of the muscular factor seems very doubtful. It has been ascertained by Mr. F. Galton, that many persons can visualise colours quite as vividly and distinctly as forms; and though no doubt individuals differ greatly here, some at least have the power of rendering colour images vivid by a prolonged effort of attention. Yet in this case there is no muscular element corresponding to that present in tracing out the contour of a particular form. That innervating the ocular muscles should react on the activity of the visualising sensory centre is, as I have allowed, quite understandable. And if this centre is at the moment in a state of sur-excitation corresponding to the idea of a particular colour, such innervation would no doubt do something to increase the excitation. But I should find it hard to believe that it could produce that very considerable augmentation of the activity of the centre, *in one direction only*, which appears to be involved in the process of reinvigorating a faint visual image by an effort of attention.

It seems to me, then, that there is room for a further supplementary hypothesis with respect to the nervous process involved in attention. Assuming what Dr. Ferrier and others

appear to be agreed upon, that attention specially involves the frontal lobes, which contain some at least of the centres for movements of the head and eyes, the question arises whether this particular region may not stand in a closer, more penetrating and more complex connection with all those regions of the brain which constitute the centres of sensation and ideation, than is involved on the theory of its being merely the centre for particular groups of movements. Wundt, it may be remembered, while fully recognising the part played by motor processes in the intensification and fixation of sensations and ideas, postulates a further nervous process, viz., a centrifugal impulse from the centre of attention or apperception to the sensorium, which is simultaneous and organically conjoined with another centrifugal process, viz., that of motor innervation issuing from the same region.

How such a process would have to be conceived it is for the physiologist to say; whether this action of the highest regulative centre on sensory centres should include an inhibitory as well as a stimulatory effect; whether it is wholly direct, or whether in part indirect, involving the control of the circulation in the cerebral capillaries through the vasomotor system—these are points on which it would ill become anybody but a trained neurologist to offer conjecture.

### *Biological Conception of Attention.*

In bringing to a close a paper which I fear may strike you as a little discursive, and already long enough, I should like to add a few words on the biological function and the practical aspect of Attention.

In the first place, then, it is evident that attention, by limiting and selecting, substitutes in our mental life a relative simplicity in place of a confusing multiplicity of elements. If, under the crowd of sensory stimuli by which we are assaulted, we had no power of selective fixation, consciousness would be nothing but a confused mass of sentience and adjustment of organism to environment would be impossible. In the highly complex life of man, as distinguished from that of the lower animals, and more particularly of civilised man,

such a power of selective reinforcement of particular elements presented, corresponding to the predominant interest of the moment, seems to be indispensable. The power of mental concentration is thus biologically correlated with the multiplex action of the environment on the human organism, and the multiplex form of reaction necessitated by this.

In the second place, all attention is in its nature temporary and self-terminating, and this not merely because we cannot indefinitely maintain the effort of attention, but because by attending now we render attention by-and-bye unnecessary. In other words, the severer exertions of mind involving exceptional expenditure of nervous energy are required only in the earlier and more difficult stages of adjustment. That this arrangement implies an economising of force is obvious.

One can understand, too, from this what is often spoken of as the special achievement of genius—the rapid mastery of a new acquisition or group of acquisitions by a supreme effort of concentration. Great mental power, which a number of writers connect with exceptional concentration, may perhaps be said to imply exceptional capacity for new adjustment through initial concentration of the most intense and vigorous kind. Such exceptional power of concentration has for its probable condition a special development of particular nervous centres, by which they become capable of frequent fits of strenuous activity.

The effect of such exceptional power of attention is to expedite the processes of intellectual assimilation, and thus to open the way for new lines of adjustive activity. Hence, as we see in the case of the greatest minds, as Goethe, the enormous range of intellectual adjustment possible. Such men, because of the splendid stock of nervous energy with which they are endowed, and also of the remarkable results of successive acts of concentration, lead the intensest lives, and are conscious in a sense compared with which the narrow stationary man of routine may be called unconscious.

Thus, the few fortunate master minds, so long as they preserve a sound digestion and other sadly-despised bodily conditions. For most men, however, what stock of con-

centrative power nature has conferred on them is apt to be limited in its direction. As was pointed out in the earlier part of this paper, attention follows the lines of interest, and for most of us interest is apt to narrow itself down, as life advances, to a few circumscribed regions. The specialization of professional work, so characteristic of the social condition of our age, compels attention itself to become the slave of that very Habit of which it is the natural enemy. The mind has to retrace again and again the same path, face again and again the same harrassing problems. And here I trust I may not seem intruding on the proper domain of the pathologist if I remind you that in this very circumstance lies one of the chief dangers to mental health. Under the pressure of fixed interests the power of attention which was developed by school discipline, and the ample indulgence of which was the chief delight of youth and early manhood, loses its alacrity, its freedom of movement. The man no longer cares to master a new subject. He listens, he reads, only as opportunity occurs to enrich his own narrow domain of ideas.

Wise men recognise the danger, and save themselves from the tyranny of paramount interest by keeping the range of interest as wide as possible. A busy professional man who can make the effort of tearing himself for the moment from daily absorbing topics and throw himself into politics, art, music, or even the lightest vein of fiction, has a safeguard against worry and other more serious evils. In view of the importance of retaining unimpaired the power of a wide impartial attention, it were well, perhaps, to direct the work of education more expressly than is done to the problem of developing this power. A boy might with advantage be exercised in concentrating on a few special subjects and at the same time in acquiring a certain knowledge of others. We seem in education, as in other things, to be drifting into extreme specialisation. The reasons are obvious. It is only by such narrow restriction of the mental gaze that exact detailed knowledge becomes possible. But it may be as well to recall what J. S. Mill said in his St. Andrew's Address, that an educated man should combine a minute

knowledge of one or a few things with a general knowledge of many things. What Mill and others have urged in the interests of a liberal culture may also be urged in the interests of mental health. To be able to take wide excursions in the field of knowledge far beyond the boundary of our own small plot is at once a sign and condition of the *mens sana* : a sign, because it shows there is as yet no morbid preoccupation with one subject ; a condition since it is by continuing to exercise this power that we shall best arm ourselves against the insinuating attacks of such preoccupation.



# ON ASSOCIATED EYE-MOVEMENTS PRODUCED BY CORTICAL FARADIZATION OF THE MONKEY'S BRAIN.

WITH A DIAGRAM.

BY F. W. MOTT, M.D., AND E. A. SCHAEFER, F.R.S.

(*From the Physiological Laboratory, University College, London.*)

OUR experiments upon the associated eye-movements which result from cortical faradization may be described under four heads, viz. :—

1. Associated movements produced by unilateral faradization of the frontal region of the cortex.

2. Effects of bilateral faradization of the frontal cortex.

3. Effects of bilateral faradization of the cortex of the occipital lobes.

4. Effects of simultaneous excitation of the occipital lobe of the one, and of the frontal lobe of the other hemisphere.

I.—*On the associated eye-movements which are caused by unilateral excitation (weak faradization) of the head and eye area of the cortex of the frontal lobe.*

Since these movements were first investigated in the monkey by Ferrier,<sup>1</sup> very little advance has been made in our knowledge of the subject. Ferrier, as is well-known, obtained, on placing his electrodes over a certain area of the frontal lobe which he has marked (12) conjugate deviation of the eyes with simultaneous movement of the head to the opposite side. Horsley and Schaefer<sup>2</sup> showed that this area must be extended to the marginal convolution above, and nearly to the lower end of the pre-central sulcus below, but otherwise obtained no constant differentiation of the eye-movements (*e.g.*, upwards or downwards as well as sideways) from excitation of the different parts of the area,

<sup>1</sup> *Proc. Roy. Soc.*, 1875.

<sup>2</sup> *Phil. Trans.*, 1888. B.

(although such differentiation is obtained on excitation of different parts of the occipital visual area.<sup>1</sup> Horsley and Beevor<sup>2</sup> subsequently made a minute investigation of the area as thus defined. They showed that the strongest lateral movements of the eyes are got by excitation in the angle of the pre-central sulcus<sup>3</sup> but otherwise for the most part failed to obtain a differentiation of the movements. Such upward inclination as they noticed was "very rarely observed," and a downward movement "was noticed but twice" in a very large number of experiments. Moreover, the points to which they localize these inclination movements do not at all correspond with the results of our experiments. Whether this difference of result is due to our having employed monkeys belonging to other genera, which is hardly likely to be the case, or to sources of error which we shall presently have to indicate, we need not, in consideration of the rarity of the results they record, here discuss.

Very early in the course of our investigations we were driven to the conclusion that it was useless to attempt a differentiation of the eye-movements unless monkeys with exceptionally large and well-developed brains were employed. These were difficult to procure, but ultimately we were successful in obtaining a number of large specimens of *Callithrix*, in which we were able to obtain the desired differentiation with comparative precision. Very definite confirmatory results were also got in one well-developed *Bonnet* monkey, and in a large *Rhesus*; whereas in smaller *Rhesus* and *Liponda* monkeys either no indications of upward and downward movements could be got, or such as were obtained were indefinite or contradictory at different periods of the experiments. This is probably to be ascribed to the fact that, at different periods of an experiment, the different parts of the head and eye region are unequally excitable, either in consequence of difference in etherization, or of

<sup>1</sup> Schaefer. *BRAIN*, April, 1888.

<sup>2</sup> *Phil. Trans.*, 1888. B.

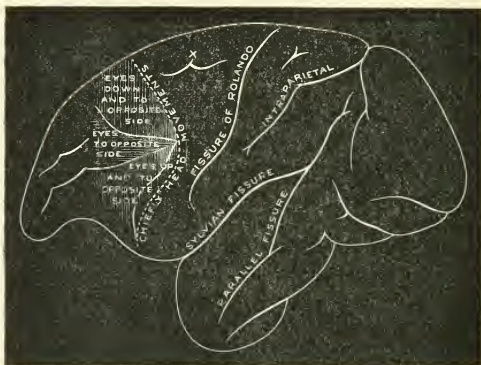
<sup>3</sup> This part of the eye area they have termed the centre of "primary representation" of the simultaneous movements of the head and eyes. We must, however, confess that we have been unable to comprehend the distinction implied by this term.

vascular disturbance consequent on exposure; and when the effect of the excitation has but a small area to spread over, it may tend to call into play those parts of the area which are at the moment more excitable. It is well-known to all who have investigated the "motor" cortex by faradization that anomalous results are frequently obtained in consequence of such increased excitability of a particular area (or of its connections in the lower or bulbo-spinal centres), *e.g.*, after a particular point has been faradized for a short time and thus thrown into a condition of increased excitability, it often happens that faradization of a neighbouring area will produce not only the effect rightly due to stimulation of that area itself, but also those which had been previously obtained from the area which had before been faradized. In this manner, if from any cause, the part of the head and eye area which is concerned with the production of upward movement of the visual axes has been rendered more excitable than that concerned with downward movement, minimal excitation of almost any part of the area, will produce upward combined with lateral movement; or *mutatis mutandis* downward, combined with lateral movement, especially if the brain be small, or the area less expanded than usual. This is one important source of error which must always be taken into consideration. Another one is to be found in the fact that, if the eyes are in the first position at the moment of excitation, and the faradization of the pia mater produces awakening from narcosis; there is then almost invariably a tendency to raise the eyes and elevate the upper eyelids, whatever be the part of the cortex stimulated. Movements of the eyelids sometimes produce the appearance of upward or downward movement of the globe when there may be only a purely lateral movement, and this is a source of error which also requires to be guarded against.

Lastly, in all experiments having for their object the determination of any upward or downward inclination combined with the conjugate lateral deviation of the visual axes, it is essential that the eyes at the moment of faradization should be in the primary position; for if they are already deflected downwards or upwards, the excitation of a spot

which would bring them from the primary position into the simple lateral secondary position, will bring them into the same (simple lateral secondary) position, and will consequently produce an upward or downward deflection combined with the lateral movement.

Some of these sources of error also complicate the investigation of the occipital (or sensory) visual area of the cortex; but this area is so greatly expanded that the difficulties are much diminished, and hence the determination of the differentiation of different movements has been far easier than in the less expanded frontal region.



The general results we have obtained from unilateral faradization of the head and eye area in *Callithrix* were demonstrated to the Ophthalmological Society on March 26th. They show that as regards conjugate deviation of the eyes this area may be regarded as consisting of three zones (see diagram), viz., (1) a middle zone immediately below the horizontal part of the pre-central sulcus, faradization of which is followed by simple lateral deviation, well-marked and without either upward or downward inclination; (2) an upper zone immediately above this, which may extend to and include part of the marginal gyrus, giving on

faradization downward inclination, usually combined with lateral deviation; and (3) a lower zone immediately below the middle one, and sometimes extending nearly to the lower margin of the hemisphere, which gives upward inclination, usually also combined with lateral deviation. When all three effects are well differentiated the zone which gives a purely lateral deviation is comparatively narrow; rarely, however, so narrow as that on the mesial surface of the occipital lobe, where zig-zagging lateral deviation is sometimes obtained, showing that it is little but a line of demarcation between upper and lower zones.<sup>1</sup> Anteriorly the middle zone tends to broaden—indeed all the zones become broader and the effects more indefinite, as the electrodes are carried forwards into the pre-frontal region.

We have not carried out observations upon the head-movements so systematically as upon the eye-movements, because these unilateral movements lay somewhat beyond the immediate scope of our enquiry, which was concerned with bilateral movements only. We have, however, observed a very definite rotation of the head to the opposite side and downwards, accompanying the downward inclination produced by excitation of the upper zone, and a rotation upwards and to the opposite side, accompanying the upward inclination produced by excitation of the lower zone, besides the simple lateral movement of the head which usually accompanies simple lateral deviation of the eyes. These head movements are most marked as the posterior limit of the head and eye area is approached; and at this limit they are frequently accompanied by movement of the arm at the shoulder, or by contraction of the muscles of the face and head. A movement of the opposite ear (pricking) is almost constantly a concomitant of the lateral deviation of head and eyes which follows excitation in the angle of the pre-central sulcus.<sup>2</sup>

Lastly, we have investigated the effects of unilateral excitation of different parts of the area after section of the corpus callosum, and after destruction of the corresponding

<sup>1</sup> We have, however, once obtained a zig-zagging lateral deviation on excitation of the middle zone of the frontal area.

<sup>2</sup> Cf. Horsley & Schaefer. *Loc. cit.*, p. 8.

area in the opposite hemisphere. Our results here have only served to confirm those of previous observers in shewing that the bilateral movements continue after as before the lesion, and that the associated effect is not necessarily produced through the corpus callosum and the cortical centres of the opposite side of the brain, but possibly through lower centres.

## II. *Effects of Bilateral Faradization of the Frontal Cortex.*

So far as we are aware, no previous observers have investigated the effects of calling into activity identical "motor" cortical centres of both hemispheres by simultaneously faradic excitation. For the ordinary limb-centres, which produce only unilateral movements, such investigation has no particular interest, but for the conjugate movements of the eyes the case is different and it would be difficult to predicate the result which is to be expected.

The method which we at first adopted was to pass the same faradic current through two pairs of electrodes, which were applied to similar points on the two frontal lobes. In this manner a stimulus of as nearly as possible the same strength is applied on the two sides, and it might be supposed that the effect produced would be equal. This is, however, rarely the case; almost invariably the action of one side preponderates over that of the other. This is due to the fact that the excitability of the two sides is scarcely ever the same, so that a minimal stimulus which would excite one side would produce no appreciable effect on the other. In order to overcome this difficulty we had to adopt a different method, viz., to employ two completely distinct induction coils and electrodes, the keys being so arranged that either one or other side of the brain could be separately stimulated, or both could be excited simultaneously. Points were in the first place found on the two sides which gave when separately stimulated exactly the same kind of conjugate deviation of the eyes (but of course in different directions), and by shifting the secondary coils, the extent and rapidity of this movement was also got as nearly as possible equal—so far as could be judged by the eye. These preliminary deter-

minations having been made, two points were, after a brief period of rest, simultaneously excited.

When two points—which give on unilateral stimulation simple lateral conjugate deviation of the eyes—are thus simultaneously bilaterally stimulated, the result is almost invariably, if the eyes are not already in the primary position, to bring them into this position and to produce fixation of the visual axes upon a distant object. Sometimes there is a slight convergence of the visual axes, and invariably, if they are previously divergent, as is often the case in narcosis, this divergence becomes obliterated and they become fixed in a parallel or slightly convergent direction. If at the moment of simultaneous bilateral excitation the eyes were exactly in the primary position, they almost invariably remain motionless. The main result therefore of equal simultaneous bilateral excitation of points, which when unilaterally excited cause simple conjugate lateral deviation, is to produce visual fixation, without any tendency to lateral deviation. If the two pairs of electrodes are placed on the upper or lower zones of the frontal area, that is to say, on similar points, which when unilaterally stimulated cause conjugate lateral deviation, combined respectively with more or less downward or upward inclination, the effect of simultaneous bilateral excitation is to cause a simple downward or upward inclination of the visual axes, as the case may be, usually with maintenance of parallelism of the axes, sometimes with slight convergence followed by fixation.

Sometimes there is a struggle between the two centres, the eyes tending first slightly in one direction and then in the other; finally, in these cases they usually tend eventually to deviate to one side. This result is, however, much more rare than those above described. Whenever an immediate lateral deviation to one side was obtained, we assumed that the bilateral excitation was unequal, and generally found that by diminishing the strength of the current which was applied to the opposite cortical centre, we could obtain the usual result. One of the most remarkable results of bilateral faradization is the modification which is frequently produced in the results of subsequent unilateral excitation.



In very many instances we have found that after even a short period of bilateral faradization, which has produced the parallelism or slight convergence and fixation of the visual axes in the manner above described, unilateral excitation does not produce the usual effect of conjugate deviation of the eyes to the opposite side, but is followed by exactly the same result as the preceding bilateral excitation, that is to say, it produces or continues the condition of visual fixation. It is, in fact, as if the lower centres had been set by the bilateral excitation in a particular groove or habit of action, from which they do not immediately return to the indifferent condition.

### III. *Effects of Bilateral Faradization of the Occipital Visual Area.*

The results obtained by us from bilateral excitation of the occipital lobes, are in all respects comparable to those yielded by stimulation of the frontal areas. Equal and simultaneous excitation of points, which give when unilaterally stimulated a simple lateral conjugate deviation, produces parallelism and fixation of the visual axes, and if the eyes are already in the primary position the stimulation is usually unaccompanied by any movement. If, however, the electrodes are placed on those zones which give, on unilateral stimulation, downward or upward inclination of the visual axes combined with lateral movement, the fixation on bilateral stimulation is then preceded by downward or upward inclination, without any lateral deviation. There may also be produced, as with the frontal area, slight convergence of the optic axes.<sup>1</sup>

### IV.—*Effect of Simultaneous Excitation of the Occipital Visual Area of the one Hemisphere and of the Frontal Area of the other Hemisphere.*

If the electrodes be placed, the one on a point of one frontal area which gives simple lateral deviation of both eyes to the opposite side, the other on a point of the opposite occipital lobe which gives the same movement of the eyes

<sup>1</sup> Schaefer, "Experiments on Electrical Excitation of the Visual Area," *BRAIN*, April, 1888. Also Obregia, *Ueber Augenbewegungen auf Schsphaerenreizung*, *Arch. f. Anat. u. Physiol., Physiol. Abth.*, 1890.



but in a contrary direction, the strength of the stimulus being so graduated as to produce in each case about the same amount of deviation when separately stimulated, then when these two points are simultaneously excited, the action of the frontal cortex invariably preponderates, so that a simple effect of lateral deviation is obtained, the same as would be produced by excitation of that frontal centre alone. To balance this preponderating action of the frontal centre it is necessary enormously to increase the strength of the stimulus which is applied to the occipital lobe. This result is in conformity with the difference of the period of latent stimulation for the frontal and occipital lobes respectively.<sup>1</sup>

<sup>1</sup> Schaefer, *International Monthly Journal of Anatomy and Physiology*, 1888.

## ON MOVEMENTS RESULTING FROM FARADIC EXCITATION OF THE CORPUS CALLOSUM IN MONKEYS.

BY F. W. MOTT, M.D. AND E. A. SCHAEFER, F.R.S.

*(From the Physiological Laboratory, University College, London.)*

IF a pair of fine electrodes, guarded except at the extremity with shellac or paraffin, are carefully passed vertically into the great longitudinal fissure between the hemispheres and are thus used to stimulate the upper surface of the corpus callosum, bilateral movements of the head, trunk and limbs are obtained according to the point to which the electrodes are applied. These movements are due to excitation of fibres in the corpus callosum and not to a spreading of the electric currents to the "motor" surface of the cortex, for (1) the effects are obtained with very weak currents, and (2) if the electrodes are somewhat withdrawn from the surface of the corpus callosum the effects immediately cease, although the excitation is now applied at a point nearer the motor surface.

The effects are obtained, roughly speaking, in the following order from before back, viz.:—When the electrodes are applied over and just behind the genu, head and eye movements are produced; further back, movements of the arms at the shoulder and of the upper part of the trunk; then movements of the forearms and general movements of the hands and fingers; then of the lower part of the trunk and tail, and lastly, movements of the lower limbs. The movements are not obtained if the anæsthesia be too deep; they become unilateral if the grey "motor" cortex of one hemisphere be destroyed by actual cautery, and if the excitation be too strong or too prolonged they pass into epileptoid contractions; they are evidently due therefore to indirect excitation of the

“motor” cortical centres. Although movements of particular parts of the body are thus roughly or generally capable of being produced by excitation of different parts of the corpus callosum the localization is not exact, for there are usually other movements produced at the same time but less markedly, as if the fibres going to the several cortical areas, although more massed at certain parts of the corpus callosum, are yet scattered over a considerable length of the commissure. This is in conformity with the degeneration results obtained in the corpus callosum by Sherrington, after unilateral destruction of localized portions of the “motor” area.<sup>1</sup>

In order to investigate more precisely the position of these fibres in the corpus callosum we have in several experiments cut away one hemisphere and directly excited the cut surface of the corpus callosum. The movements then obtained are of course confined to the side of the body which is in nervous connection with the intact hemisphere, that is to say, if the left hemisphere be cut away, the movements are on the left side of the body.

Operating in this manner we have been able to obtain much more distinct and specialized movements than when the intact commissure is stimulated at the bottom of the longitudinal fissure, but the experiment is far more difficult to perform in this way, and is frequently entirely vitiated by the profuse hæmorrhage which usually follows the removal. The general results are, however, the same. Movements of the eyes and head are got most anteriorly, of the lower limb most posteriorly; and of the upper limb and trunk between. With the weakest possible excitation, a favourable slight condition of narcosis, and a freshly-made section of the commissure we have occasionally obtained very precisely specialized movements, such as a simple conjugate lateral deviation of the eyes, simple opposition of the hallux and the like. These simple movements were, however, rarely got; usually although one part showed a greater tendency to move, other and even distant parts of the body might also but less strongly be set in action, thus indicating as before, a scattering of the commissural fibres. Even when the movements are confined

<sup>1</sup> *Journal of Physiology*, vol. x.

to one part of the body or one limb, they are very seldom completely specialized, but are usually general movements. This is the case, not only when the cut surface is stimulated, but as might be supposed also when the excitation is applied from above to the intact corpus callosum. For instance, the flexors and extensors of a limb are often both thrown simultaneously into action, and the eye-movements instead of being definite movements of conjugate deviation are frequently indefinite or rolling movements. Nor, in the case of the stimulation of the intact corpus callosum, are the muscles which are called into action always quite the same on the two sides of the body, although those which are most strongly affected always belong to the same part. This may easily be understood, if we consider that the bundles of fibres which pass within the corpus callosum to corresponding points in the grey cortex of the hemispheres would not necessarily cross at exactly the same point, for those passing to the one side might be placed more superficially or more anteriorly as compared with those passing to the other side: hence we should expect to find some differences in the movements of the two sides.

For some reason which we are unable to explain, we were never successful in obtaining contractions of the muscles of the face from stimulation of any part of the commissure.<sup>1</sup>

It is not improbable, however, that the commissural fibres for their motor centres (and perhaps also for those of the larynx) may yet be found in the rostrum of the corpus callosum. We certainly explored this part on two or three occasions, but the negative results we obtained may perhaps be ascribed to the hæmorrhage which accompanied its exposure. This may also account for the fact that we failed to get any movements on stimulating the splenium, although we had expected to obtain movements of the eyes from its excitation. Practically all the results which we have obtained have been yielded by the thinner middle part or body of the great commissure. We have performed a con-

<sup>1</sup> We were also unsuccessful in obtaining any independent movements of the fingers, although we once or twice obtained such movements of the toes.

siderable number of experiments and have employed for the purpose several different kinds of monkey (*Liponda*, *Rhoesus*, *Bonnet*, *Callithrix*). The most precise evidence of localization within the commissure which we obtained in any was got from a fine, intelligent *Bonnet*; but the conditions of experiment may have been accidentally favourable on this occasion. The sequence of movements obtained by stimulating the cut surface from before back was very distinct in this case and certain movements were easily isolable. In most other animals the movements were, as has been above mentioned, of mixed character, with occasional preponderance of movement of particular parts. Thus in one case retraction of the arm and shoulder, combined with flexion of the leg and hip, recurred as a prevalent action. In another case, in which we stimulated with flat paraffin-guarded electrodes successive points of the under surface of the corpus callosum, we obtained along the whole length of the excitable part, movements of the eyes and eyelids.

The general results of our experiments and the inferences which may be drawn from them are briefly the following:—

1. The corpus callosum contains fibres excitation of which produces movements on both sides of the body.

2. These fibres do not pass directly from the corpus callosum to the internal capsule<sup>1</sup> but enter the grey cortex of the hemispheres, and there come into connection with the so-called “motor centres.”

The fibres which pass to the several motor centres, although mainly massed in definite parts of the corpus callosum, are not confined entirely to those parts, but are to a certain extent scattered along the middle thinner part of the commissure.

<sup>1</sup> When destruction of the grey cortex was effected by actual cautery, as before described, care was taken to avoid injuring any connection between the corpus callosum and the internal capsule.

# A CONTRIBUTION TO THE STUDY OF CHEYNE-STOKES BREATHING.

BY J. DIXON MANN, M.D., F.R.C.P.,

*Physician to the Salford Royal Hospital. President of the Manchester  
Pathological Society.*

THE phenomena comprised under the designation Cheyne-Stokes breathing, in a comparatively short time have called forth a copious literature on the subject, derived both from experimental and from clinical observations. Many hypotheses have been advanced explanatory of the periodic alterations in breathing, but as yet without general acceptance being accorded to any. I have recently had under continuous observation a case, probably unique, in which Cheyne-Stokes breathing lasted for more than a year. It afforded an admirable opportunity of studying the symptoms during life, and of searching after death for pathological changes affecting the organs concerned in their production. Since the subject is involved in obscurity, it is possible that symptoms which at present appear to have no relation to the phenomenon in question, may in the future aid in its interpretation, I therefore present, in as few words as possible, a full report of the case.

The patient at the time of his death was seventy years of age. He never enjoyed robust health, but, until the latter period of his life, he never had any illness of importance. There is nothing in the family history that has any bearing on the case. The patient was very careful and abstemious in his habits. He was endowed with the highest type of mental capacity, and until the age of fifty he had exercised his mental powers to the fullest extent in original scientific investigations, necessitating much profound thought and elaborate calculations. My first professional relation with

him was in 1874. Previous to this time, and for several years after, he suffered from attacks of profuse epistaxis. For two years his general health had gradually deteriorated without any decided symptoms, and he became depressed in spirits, displaying much anxiety about his condition; afterwards he became more cheerful. About this time, the patient used to complain of an uncomfortable sensation at the base of the brain, which he localised by opening his mouth and pointing with his finger to the posterior part of the palate, explaining that the sensation was inside the head. Still later he suffered from a feeling of confusion in the head, with some amount of pain in the parietal regions, which, however, was not violent.

In 1880, he developed a soft mitral systolic murmur, which could not be heard beyond the anterior axillary fold. The left ventricle was subsequently somewhat hypertrophied, but the apex beat never descended more than half an inch below the normal position. The enlargement was much less than might have been expected from the atheromatous condition of the arteries found after death. The heart beats were always very regular; the pulse never partook of the character of the mitral pulse, neither in smallness nor in intermission. As will be mentioned later on, there was an occasional intermission, but it was accidental rather than habitual. The condition of the heart never affected the breathing, the regurgitation evidently being slight. The lungs were healthy, the breathing powers being excellent. The patient never suffered from any bronchial affection.

In 1887 he had an attack of partial paralysis of the right arm and leg, with transient motor aphasia. In a few weeks the paralysis almost disappeared. The aphasia passed off in a few days, but the speech remained less distinct than before. The mental powers had been gradually diminishing, and their downward progress was hastened after this attack. The epistaxis had ceased to recur for some time before the paralysis, and never returned. On August the 5th, 1888, after a considerable amount of cerebral excitement, which came on without external cause, he entirely lost the use of the right side. The hemiplegia was complete, and included

the lower part of the face on the right side. The tongue on protrusion was pushed over to the paralysed side, the right angle of the mouth dropped somewhat, and the masseter of the same side was flabby. The upper facial muscles were unaffected. He could without difficulty eat food which had been prepared by being finely divided, but he had little power of mastication. So far as could be made out, the organs of special sense were not affected. This attack was accompanied by complete motor aphasia, which was permanent, the patient never regained the power of speech. On account of the aphasia and the failure in the mental powers, no reliable evidence could be obtained as to sensory disturbance. In a few hours after the attack, some amount of motility returned in the leg, the arm remaining useless.

On August 19th, 1888, he began to breathe irregularly, and, during my absence from home, he was seen by my colleague, Dr. Edge, who found he had well-marked Cheyne-Stokes breathing. On my return, two days after, his condition was much as before, with the addition of the Cheyne-Stokes breathing, which continued. In a few weeks the general condition improved, and he became sufficiently strong as to walk unaided; the arm also improved, but never recovered much power of movement. The condition at this period and for some time afterwards was as follows:—Pulse from 64 to 72, regular and not excessive in tension; appetite excellent; bowels obstinate, requiring constant attention; sleeps well; skin dry and cold, there was never excessive perspiration. Sensation appeared unaffected throughout. There was no special atrophy, although the muscles generally were wasted. There was no rigidity. The knee-jerk was slightly increased for a time on the right side, afterwards was abolished on both sides. The skin reflexes were early lost on the right side. The left leg was often moved about for hours together when the patient was in bed, apparently as an outlet for cerebral irritability. The urine, usually amounting to 40 to 60 ounces in the twenty-four hours, was of medium colour, free from deposit, s.g. 1,010 to 1,016, no albumen, urea from 350 to 385 grains in the twenty-four hours. On several occasions a considerable excess of chromogen was present in



the urine, probably indol, with some other substance, the oxidised product of which was not soluble in ether. When the urine was treated with hydrochloric acid and bleaching powder it yielded a claret-colour, and on being shaken out with ether, imparted a blue which gave the absorption spectrum of indigo-blue. No relation could be traced between this condition of the urine and any symptoms present at the time. There was never any dropsy, nor even œdema of the ankles. The bladder was unaffected. The patient could protrude the tongue to the full extent, could masticate feebly, and could swallow both solid and liquid food without difficulty. There was never any tremor of the tongue, nor of the lips, nor hands. At one time bulbar symptoms seemed imminent, but did not advance beyond some difficulty in swallowing, necessitating the food being introduced to the back of the mouth. The bulbar symptoms disappeared in about a week. The only vocal sounds the patient could produce were "ai" and "o," meaning yes and no. He could not read, but liked to look at the pictorial papers. He had occasional attacks of sickness, which resembled "bilious attacks" more than gastric crises. During all this time the mental condition progressively deteriorated, paroxysms of restlessness and irritability occasionally manifesting themselves. With slight fluctuations, the physical condition underwent no perceptible change for several months. The patient was dressed each day, and with a little assistance walked up and down stairs; he sat to table when eating his dinner, the nurse feeding him. At the earlier part of the illness he wished to have the newspaper read to him, but it was doubtful how far he comprehended the subject-matter. Later on he quite failed to take interest in anything, and only afforded a momentary and partial recognition to his relatives. On August the 17th, 1889, he had an attack of syncope. On September the 9th the back became very weak and he could not erect himself as formerly; at this date he ceased to be able to walk. On October the 10th he became cyanotic for a few minutes; when seen two hours after, the pulse was quickened and the cyanosis had disappeared, the patient being very prostrate and in a semi-comatose con-

dition. The following morning the coma had deepened and the pulse was running down. Death took place at 9.45 p.m. on the same day.

The chief medical interest in the case centres in the Cheyne-Stokes breathing and its prolonged duration. I will therefore give a detailed account of the phenomenon :—

The periodic breathing was unaffected by position. Having visited the patient at weekly intervals during the whole time that he suffered from the Cheyne-Stokes breathing, I had abundant opportunity of observation under a variety of conditions, as when lying in bed on his back, on the right and left sides, sitting propped up in bed, sitting on chair, and also standing upright. The character of the phenomena was precisely the same in all cases, allowing for variations in the duration of the phases of which each cycle was composed. Such variations were not dependent on the position of the body, nor on any other external condition, but occurred from time to time in the progress of the disease, and lasted from a few hours, to days or weeks. The following description is from notes taken at the time, after close observation of the symptoms for many weeks. The patient is sat up in bed with the legs resting horizontally before him. The chest is covered with night shirt only, and the lower part of the body and the legs with a sheet and counterpane, so that any movement can readily be seen. The trunk is upright, the arms resting on the bed clothes covering the thighs, but not supporting (or only to a slight extent) the upper part of the body, which is maintained in position partly by pillows placed behind it, and partly by the dorsal muscles. The face is somewhat pallid—not anæmic in the sense of being entirely devoid of colour, but like that of a person who has been confined to his room for a few months. The head is slightly bent forwards, as would be the case if the patient was looking at his feet, the line of sight being at a right angle to the plane of the face. The right side of the face being partially paralysed, displays the corresponding angle of the mouth at a lower level than that of the opposite side; the lips and teeth are separated to a slight extent. The lips are normal in colour. The eyes are open so that the pupils can be clearly seen. The

pupils are about two millimetres in diameter, they are equal, and respond both to light and to accommodation. Taking the cycle in the midst of the respiratory phase, the patient is seen to be breathing in a perfectly normal manner. After a few respirations, the depth of each successively diminishes, and after six more the breathing has entirely ceased. These six downward steps are not equal: if represented graphically, a line drawn along their apices would form a curve, showing that the diminution is proportionally greater in the first three than in the last three. The time occupied by each respiratory act during the downward progress is the same as that required for each act of tranquil respiration, the height of the waves diminishes, but the intervals between them are unaltered. During the pause the face is perfectly still; the eyes remain open, and there is a longer interval than usual between the winking of the lids. The facial expression is peculiar: it is that of a person absorbed in thought, as when the eyes are fixed on an object of which the mind is not cognisant, the brain being entirely occupied in profound cogitation on a subject far removed from the physical surroundings. Such is an exact description of the facial expression, but as a matter of fact no cerebration is taking place. The pupils remain unchanged; in no single instance could I detect any diminution in their size during the pause. The colour of the face, including the lips, is unaltered, there is not the slightest appearance of cyanosis nor of flushing. The ends of the fingers under the nails also retain their colour. If left to himself the patient is usually perfectly still during the pause, but he is not unconscious. If I say—"Well! good morning," and hold out my hand as though to shake hands before leaving, he makes a movement of the hand as one who is wishful to reciprocate the salutation; if requested to put out the tongue he does so. To obtain response, however, it is necessary to utter the words in a louder tone than customary. The movement of the hand is not accompanied by any change of facial expression, nor in the direction of the eyes, as is the case under similar conditions when respiration is taking place. It has all the appearance of an automatic act, the words provoking the movement without the intervention of volition. No

artificial stimulus had any effect on the duration of the pauses. The pulse is not altered neither in rate nor in character during the pause. This was ascertained on many occasions, both by the finger and by the sphygmograph. The duration of the phenomena enabled me to make numerous observations which were carried out with the greatest care, the important bearing of this question being obvious. Tracings were taken during entire cycles, without revealing the slightest alteration, either in rapidity or in tension. There was an occasional irregularity in the pulse-rate—rarely amounting to intermission—but the inequality was limited to individual cardiac contractions and in no instance had it any relation to the respiratory function. After an interval of total cessation of breathing, usually of from 15 to 30 seconds in duration, it is re-established by a series of respirations, of which the first is exceedingly shallow, the second deeper, and so on until the normal depth is slightly exceeded. Then follow three or perhaps four respirations of this slightly exaggerated type, after which the respirations become tranquil and easy until the cycle recommences. The breathing rate during the phase of tranquil respiration was usually about 18 to the minute. During the period of forced breathing the right side of the thorax did not expand so freely as the left. The duration of the period of breathing, taken from the first respiration after a pause to the last which preceded the following pause usually varied from 20 to 42 seconds. The period of breathing did not consist of a series of respirations forming an upward and downward curve, the latter immediately following the former, but the two were separated by a horizontal line representing a phase of regular and equal breathing.

When breathing was resumed, the patient's face lost the look of abstraction, otherwise there was no change. As previously mentioned, the pupils did not contract during the pause, nor was there any change in size on return of breathing. On no occasion did I see the least trace of involuntary movement of the limbs, nor twitching of the mouth when the breathing recommenced, nor was there any rigidity of the muscles of the limbs during the pause. When awake, in addition to voluntary movements of the eyes, the patient

usually made some slight change of position after the breathing was re-established, this was evidently to relieve the irksomeness of the previous fixity of position. The stillness during the pause only occurred when the patient was unoccupied at the moment of cessation of respiration. If he was walking at the time, even going up or down stairs, he continued to do so. It not unfrequently happened, after leaving his room and arriving in the dining room, that he sat down just after the pause commenced; the absence of breathing then appeared even more remarkable than at other times. After exercise the succeeding forced respirations were more energetic than usual and continued during the entire respiratory phase. If the patient was eating he continued to masticate and to swallow the food during the pause just as at other times. On these occasions the facial expression conveyed the impression that the movements, whether of walking or of eating, were non-volitional. At intervals during the last few months of his illness, the patient suffered from attacks of excessive secretion of mucus into the larger bronchial tubes. These attacks came on quite suddenly and without external influences, such as cause bronchial catarrh. They were apparently due to abnormal action of the nerves presiding over the bronchial secretion, and they disappeared as suddenly as they appeared. When this excess of mucus was present, the breathing was much embarrassed. On account of the hemiplegia, together with the general feebleness, the patient had very little power to cough, the mucus consequently accumulated until suffocation seemed imminent. At such times the forced respirations which followed each pause were dyspnoeal in character, the expiratory efforts being excessively energetic; they entirely displaced the phase of tranquil breathing and continued until the commencement of the descending steps which led to the succeeding pause. On these occasions the periods of breathing were shorter than usual. The face became distinctly cyanosed, and the patient was much distressed; this he manifested at each re-establishment of breathing by beating the bed clothes with his hands, and by groans and loud cries. During the pauses, when suffering from these bronchial attacks, he remained perfectly

still as at other times, the contrast between the two phases being most remarkable.

The phases of which each cycle was composed were fairly constant for any one period, but they underwent certain changes at various stages of the disease. At the early period of the Cheyne-Stokes breathing there was at times no actual pause, the curve representing the gradual lessening in amplitude of the respirations no sooner approached the base line than it began to rise again. This lasted only a few days, and, with one or two exceptions of not more than two or three hours' duration, did not return. The shortest pause observed by me lasted eight seconds, and the longest thirty-eight seconds. I was assured by the nurse that many times during sleep the pauses lasted much longer than the longest of these two periods, so long that, although she was accustomed to the periodic breathing, she felt convinced on several occasions the patient "had drawn his last breath." The shortest period of breathing observed by me was seventeen seconds, and the longest sixty-one seconds. On one occasion (May 13th, 1889) the breathing was continuous during sleep, but resumed the periodic type when the patient awoke. Again, on August 11th, 1889, the breathing was continuous for several hours both sleeping and waking. With these two exceptions, the Cheyne-Stokes breathing continued from August 19th, 1888, until October 11th, 1889, a period of 417 days. About twelve hours before death the periodic breathing ceased and was replaced by continuous laboured breathing, such as frequently precedes death from cerebral disease. This lasted six hours, after which the Cheyne-Stokes breathing was resumed and continued until death took place. About an hour before death the patient yawned repeatedly with extreme energy, so much so that the nurse thought it necessary to support the lower jaw with her hand to prevent dislocation. No treatment, nor drugs, as amyl nitrite, pilocarpine, strychnine, &c., had the least effect on the Cheyne-Stokes breathing. I am indebted to my friend, Dr. Robinson, of Owens College, for the following account of the pathological changes which he found in the brain and medulla.

The most important changes which were noted when the

macroscopical examination of the brain was made related to the arterial system: the vertebral, the basilar, and all the cerebral arteries and their branches had undergone a degeneration which converted them, almost entirely, into calcareous tubes.

The membranes were adherent to each other over the vertex of the brain, and over the same region the dura mater was almost inseparable from the skull-cap, but the membranes were not adherent to the cerebral substance.

Macroscopical sections of the cerebral hemispheres revealed no degenerative changes, but all the cavities of the brain, especially the lateral ventricles, were markedly dilated.

The medulla and the pons were hardened in Müller's fluid, and the former was afterwards embedded in celloidin and cut into sections, from a short distance below the calamus scriptorius up to the acoustic tubercle. Microscopical examination of these sections, after staining by Weigert's and Pal's methods, revealed two distinct abnormalities. First, there was well-marked degeneration of one pyramidal tract; and second, there was very distinct capillary congestion throughout the regions of the root nuclei of the spinal accessory and vagus nerves, the crowding of the corpuscles in the capillaries of these regions contrasting strongly with the absence of any such appearance in the surrounding areas.

Space will only permit mention of a few out of the many theories advanced in explanation of the mechanism of Cheyne-Stokes breathing. They may be divided into two groups; those which are, and those which are not based upon an assumed close correlation between the symptoms and the chemical condition of the blood. Of the first group, the earliest explanation was that of Traube,<sup>1</sup> who postulated a diminished irritability of the respiratory centre from cerebral pressure, or from deficient or deteriorated blood supply. This necessitated more carbon dioxide than in the normal condition to stimulate the centre into activity, and therefore, until the requisite amount accumulated the respiratory centre was inactive. When sufficient carbon dioxide accumulated, the endings of the vagi in the lungs were first stimulated, producing the shallow

<sup>1</sup> *Berlin. klin. Wochenschr.*, 1874.



breathing which immediately follows the pause. Next, the cutaneous nerves were stimulated, causing the stage of laborious breathing. The deep breathing soon relieved the blood of its excess of carbon dioxide, and then the stimulation of the respiratory centre subsided in inverse order—that from the cutaneous nerves first, afterwards that from the vagi. Traube subsequently added exhaustion of the respiratory centre as an additional factor in the causation of the pause.

Filehne<sup>1</sup> amplified this theory by supposing that the vaso-motor centre as well as the respiratory centre is implicated, and that in the normal condition venous blood stimulates first the respiratory centre, and afterwards the vaso-motor centre. He explains the phenomena thus:—the irritability of the respiratory centre is diminished, and it consequently fails to respond to the stimulus of ordinary venous blood. During the pause the blood increases in venosity, but on account of lessened irritability the respiratory centre is not stimulated into activity until the vaso-motor centre responding to the excessive venosity of the blood produces contraction of the arterioles and so lessens the peripheral blood-supply. Along with other parts the respiratory centre suffers from the anæmia thus caused, and is thereby stimulated into activity. Then follows the shallow breathing which deepens into dyspnoea before the arterioles have time to regain their normal condition and permit the now arterialized blood to reach the medulla. Filehne instances increased arterial tension during, or even before the pause, in corroboration of his hypothesis. Filehne's theory in its turn has been ingeniously amplified by Byrom Bramwell.<sup>2</sup>

We now come to the second group. Mosso<sup>3</sup> regards Cheyne-Stokes breathing merely as an exaggerated form of a physiological phenomenon which may be observed during normal sleep in the human subject, especially in advanced age, and also, as observed by Luciani and Fano,<sup>4</sup> in the sleep of hibernating animals.

<sup>1</sup> *Berlin. klin. Wochenschr.*, 1874.

<sup>2</sup> *Diseases of the Heart and Thoracic Aorta*, Edinburgh, 1884.

<sup>3</sup> *Archiv f. Physiologie*, 1886.

<sup>4</sup> *Lo Sperimentale*, 1879 & 1883.



Rosenbach<sup>1</sup> regards lessened irritability of the central organs of the nervous system especially of the respiratory centre in the medulla, due to defective nutrition from diminished or altered blood-supply, as the causal factor in the production of Cheyne-Stokes breathing. The centres not only have their irritability lowered, but are also more easily exhausted by performance of function. He states that neither artificial respiration, nor the inhalation of oxygen exercise any influence on the periodic breathing.

Quite recently Wertheimer<sup>2</sup> has advanced the hypothesis that the diminished irritability of the respiratory centre, which is the primary cause of Cheyne-Stokes breathing, is due, in some cases, to purely dynamic influence—to an incomplete inhibition. He expresses the opinion that various lesions of the cerebrum, either by nerve-conduction or by modification of the intra-cranial pressure, produce irritation of the medulla, and thus determine Cheyne-Stokes breathing.

In discussing the probable ways in which the respiratory movements may be modified by abnormal influences on the respiratory centre, it will be necessary first to review as concisely as possible the recent physiology of the subject.

According to Marckwald<sup>3</sup> there are in the medulla, in close relation to the nucleus of the vagus, double respiratory centres—an inspiratory, and a less easily excitable expiratory centre, the former of which only is called into action in normal respiration. There are no higher centres for respiration. The respiratory centres in the medulla are both automatically active and reflexly excitable, but, automatically, they can only occasion spasmodic, not rhythmic breathing. Normal rhythmic respiration is a reflex act principally called forth by the intervention of the vagi, which prevent abnormal accumulation of tension in the centres, and convert the inherent irritability of the respiratory centres into regular movements. The vagi have a tonus which probably is sufficient, when the organism is at rest, to regulate the respiratory movements. Next to the vagi, the upper cerebral tracts exercise an important function, and are capable of

<sup>1</sup> *Zeitschr. f. klin. Med.*, 1880.

<sup>2</sup> *Archives de Physiologie*, 1890.

<sup>3</sup> *Zeitschr. f. Biologie*, 1886.

replacing the vagi, as they (the vagi) are also capable of compensating for the loss of the upper tracts. If a portion of the upper tracts fail the vagi continuing to act, Cheyne-Stokes breathing may occur. For the production of periodic breathing no alteration in the irritability of the respiratory centre itself is necessary. The centre, either during the period of breathing or during the pause, can be equally excited by similar weak stimuli. The normal irritability of the respiratory centre is not dependent either on want of oxygen, nor on excess of carbon dioxide in the blood. The condition of apnœa has nothing to do with the gaseous contents of the blood, but is probably caused by liberation of the accumulated energy of the respiratory centre by the action of the vagi. During the apnœa neither direct excitation of the medulla, nor of the vagi can produce respiratory movements. On the other hand, Langendorff and Franck,<sup>1</sup> from experimental observations, came to the conclusion that the respiratory centre is not only automatically active, but that it can itself maintain rhythmic breathing, which differs but slightly from ordinary respiration. Loewy<sup>2</sup> made a series of experiments on rabbits to ascertain the influence exercised by the upper brain tracts, the vagi, and other nerves, on the respiratory centre. When the respiratory centre was separated from the higher centres by cutting through the medulla immediately above the respiratory centre, very little alteration was produced in the breathing. When the vagi alone were divided the breathing was slowed and deepened without being materially altered. When both upper tracts and vagi were detached from the respiratory centre a marked change of rhythm occurred. Contrary to Marckwald, but in accord with Langendorff, Loewy holds that the respiratory centre is capable of liberating rhythmic impulses automatically. In two of Loewy's experiments, Cheyne-Stokes breathing occurred after detachment of the upper tract, but when the vagi were subsequently divided the breathing regained the normal rhythm. Wertheimer<sup>3</sup> found on dividing the cord in

<sup>1</sup> *Archiv f. Physiologie*, 1888.

<sup>2</sup> *Pflüger's Archiv*, 1888.

<sup>3</sup> *Loc. cit.*

dogs, immediately below the medulla, and then performing artificial respiration until automatic respiration returned, that the breathing was of the periodic type. This points to the existence of sub-centres in the cord below the medulla, which may act automatically. He therefore would not limit the area affected in Cheyne-Stokes breathing to a circumscribed portion of the central nervous system, but would distribute it over the whole extent of the grey axis which governs the respiratory mechanism. He found that Cheyne-Stokes breathing is not necessarily the result of changes in the gaseous condition of the blood, nor need there be any alteration in arterial tension. Some recent experiments of Geppert and Zuntz<sup>1</sup> tend to prove that abnormal balance of oxygen and carbon dioxide in the blood is not the only factor by which it may act as a stimulant to the respiratory centre. By detaching the muscles of a limb in a rabbit from all nerve-paths which normally connect them with the medulla, and then tetanising the muscles, any action produced on the respiratory centre can only be transmitted by the blood. The effect produced was increased respiration, such as results from voluntary active exercise of the muscles. It was found by direct analysis that the tendency to excessive venosity of the blood from the increased muscular activity was more than compensated for by increase in the respiratory acts—that the blood was more aëriated than was necessary to maintain the gaseous equivalent that occurs in rest and work under normal conditions. The inference is that certain non-gaseous substances in the blood, resulting from tissue-change, are able directly to stimulate the respiratory centre without the intervention of the endings of the vagi. In relation to this question, Lehmann<sup>2</sup> found by experiments that diminished alkalinity of the blood, caused by the acid products resulting from muscular metabolism, is a very important factor in the stimulation of the respiratory centre. He conjectures that possibly the effect of carbon dioxide on the respiratory centre depends on its acid properties.

It will be seen from some of the experiments that the

<sup>1</sup> *Pflüger's Archiv*, 1888.

<sup>2</sup> *Pflüger's Archiv*, 1888.

older view—that of Flourens—which regards the respiratory centre in the medulla as the prime and sole mover in the respiratory act, is no longer tenable. There is abundant evidence to prove that there are sub-centres in the cord below the medulla, and there is reason to believe that these sub-centres are competent to liberate respiratory impulses of modified character without the aid of the medullary centre. As expressed by Langendorff,<sup>1</sup> the automatic centre is not an anatomical, but only a physiological unit, which is made up of the conjoint action of the centres in the medulla and in the cord, its activity being modified by the regulating centres in the medulla. For the present purpose it will be unnecessary to discuss the duplex function of the medullary centres.

Let us now consider by the light of the above-named experiments and others presently to be mentioned, together with the results of my observations on the case narrated, the various hypotheses advanced in explanation of the phenomena of Cheyne-Stokes breathing.

The theories of Traube and Filehne are based on a supposed failure of the respiratory centre to respond to the stimulus of ordinary venous blood, and that an excessive accumulation of carbon dioxide is required to bring it into action. They differ as to the mechanism by which this is effected. Against Traube's theory is its failure to account for the gradual increase in amplitude of the respiratory movements after each pause. If the renewed activity is the result of excess of carbon dioxide in the blood, this stimulus will be at the maximum immediately before the first respiratory movement after a pause. With each subsequent respiration the amount of carbon dioxide would be lessened and its power over the respiratory centre consequently diminished; but notwithstanding this the activity of the centre goes on increasing progressively, and the respirations for a time continue to augment in amplitude. To overcome this objection, Filehne makes the excess of carbon dioxide act on the respiratory centre through the intervention of the vaso-motor centre, and in this way gains time for the gradual

<sup>1</sup> *Arch., f. Physiologie*, 1887.

development of the respiratory movements. One of the principal clinical objections to this theory is the fact that increase in arterial tension—necessitated by the theory—is by no means invariably present in Cheyne-Stokes breathing. Since the theory was formulated a great many cases have been recorded in which no alteration in tension occurred. My case adds another to the list. In many cases in which alteration of tension has been observed, no constant relation existed between the changes in tension and the phases of periodic breathing. In some the tension was greatest at the end of the pause; in others during the phase of the greatest amplitude of respiration, or at the period of maximum dyspnoea. Rosenbach directs attention to the want of accord between the regular alternations of the respiratory phenomena and the capricious behaviour of the circulatory changes, as evidenced by clinical observations. Such discrepancies make it impossible to regard any alteration in arterial tension that may occur as a coefficient in the production of Cheyne-Stokes breathing. Physiological experiments also are against this hypothesis. Sokolow and Luchsinger,<sup>1</sup> and independently of these observers, Langendorff and Siebert,<sup>2</sup> have shown in frogs, that when the circulation through the medulla is prevented, either by clamping the aorta, or by rapid bleeding, periodic respiration is produced. If after clamping the aorta and production of periodic breathing the clamp is removed, the normal type of respiration returns after a varying period, which has a direct relation to the length of time that the circulation through the aorta was interrupted. All these observers agree in attributing the periodic breathing to exhaustion of the respiratory centre. Filehne suggests that if exhaustion is the cause it ought to assert itself after each individual act of respiration. Luchsinger's explanation is, that the irritability of a nerve centre increases when after rest it is again stimulated, and diminishes if the period of activity is prolonged. Although the stimulation may be continuous, the effect of the stimulation may manifest itself in periodic impulses. In illustration of

<sup>1</sup> *Pflüger's Archiv*, 1880.

<sup>2</sup> *Archiv f. Physiologie*, 1881.

this he cites the experiment by Stirling<sup>1</sup> of administering weak rhythmic induction waves to the foot of a frog, by which a series of increasing and decreasing contractions followed by a pause were obtained. Langendorff maintains that the exhaustion itself is solely antagonistic to the renewal of rhythmic breathing, and that its disappearance is gradual. Therefore the first movement after a pause being still under the influence of the subsiding exhaustion, is feeble. Each succeeding breath is further removed from the antagonistic influence, and consequently increases in depth until full amplitude of movement is attained. The movements then diminish, because the energy expended is greatly in excess of the supply. In another experiment, Sokolow and Luchsinger measured the blood-pressure in a frog by means of a monometer, after the production of Cheyne-Stokes breathing by clamping the aorta (the aorta being subsequently released), without any material change being observed; the integrity of the vaso-motor apparatus was demonstrated by stimulation of the sensory nerves, which occasioned marked increase in the blood-pressure.

In my case, it is to be noted that when the patient was in his ordinary condition there was no appearance of cyanosis during the pauses, however long they lasted. When the gaseous interchange in the pulmonary air-vesicles was impeded by excess of mucus in the bronchial tubes, however, there was marked and continuous cyanosis, and during each period of breathing the patient displayed all the anxiety of one suffering from impending asphyxia. Granting that diminished irritability of the respiratory centre is the cause of the cessation of breathing, and that excess of carbon dioxide is the cause of its periodic renewal, the pauses ought to have been abolished so long as this state of cyanosis lasted, since the venosity of the blood then existing was undoubtedly greatly in excess of that which occurred in any period of respiration when no excess of mucus was present. If the amount of carbon dioxide in the blood during the ordinary course of the Cheyne-Stokes breathing was sufficient to stimulate the

<sup>1</sup> *Sächsische Berichte*, 1874.

sluggish centre for a time, the vastly increased amount under the conditions indicated would have been sufficient to have kept it in activity as long as the excess continued. It may be urged that the respiratory centre adapted itself to the new state of affairs, its irritability being further reduced, so that its relation to the increased venosity of the blood was the same as before, the entire level at which the phenomena took place being lowered. There is a limit, however, to the depression in irritability of centres primarily concerned in the performance of functions necessary to the maintenance of life. Beyond a certain point it is no longer a question of susceptibility to the action of stimuli only that is concerned; that is simply one manifestation of an essential condition—the capacity of the elements involved to liberate energy. When the irritability of the respiratory centre is excessively lowered by faulty nutrition, so is its energising capacity—the former being the outcome of the latter, or rather, both are dependent on the same molecular activity. If the molecular activity is lowered to such a degree as to necessitate an ever-increasing amount of stimulus to compel liberation of energy, it constitutes proof of a progressively diminishing capacity of the cells implicated to yield energy. When the bronchial secretion was in excess, the venosity of the blood was so great that, had it been simply a question of reduced irritability of the respiratory centre—to any degree compatible with retention of molecular activity equal to performance of function—it must have been stimulated into continuous activity. If, as suggested, the level of the phenomena had been lowered proportionately to the excessive venosity of the blood during the bronchial attacks, the energising power of the respiratory centres would have been incompetent to liberate a series of effective impulses; the most that they could have done would have been to yield detached impulses at irregular intervals—the outbursts of final exhaustion—and that for a short time only. A further objection is, that depression in irritability such as enables nerve centres to accommodate themselves to altered external relations is of gradual development, and is not suddenly produced by excess of stimulus. The effects of exhaustion



from over-stimulation do not at present enter into consideration.

The case itself afforded further and positive proof that the immediately antecedent condition of the blood was not concerned in the production of the Cheyne-Stokes breathing. When a pause occurred whilst the patient was walking from one room to the other (the bronchial secretion being normal), the breathing during the subsequent period of respiration was dyspnoeal throughout, the excessive amplitude of movement not being limited as under ordinary conditions to the first two or three breaths. This points to actual stimulation of the respiratory centre, probably to be explained by the light of the previously-mentioned experiments of Zuntz and Geppert, as resulting from the non-gaseous products of muscular activity, there being no appearance of cyanosis on such occasions. Nevertheless, there was no interruption to the Cheyne-Stokes breathing. Although evidently undergoing excessive stimulation and responding to it, the respiratory centre ceased to do so in the midst of the excitation. When the patient sat down further development of these respiratory stimuli from muscular movements ceased, but their elimination was not completed during the following period of breathing; it was resumed after the first, and sometimes after the second pause, in the succeeding respiratory phases. Under these circumstances, there is no time for the respiratory centre to adapt itself to altered conditions, the stimulus is developed in a few seconds, and produces an immediate effect. Further development is suddenly arrested. The limited period of respiration which follows is insufficient for the elimination of the stimulus, and instead of the period being prolonged until all excess is removed, and the balance between the respiratory centre and the blood restored (as would be the case if the intermission depended on ineffective stimulation of the centre), the completion of the task is held over till after the next pause.

It is obvious that a causation other than the chemical condition of the blood must be sought for, and the attention is naturally directed to changes in the respiratory centre itself. In the normal condition we may suppose that the



nerve cells concerned in the production of the respiratory movements convert the oxygen and other substances contained in arterial blood, which are spoken of as nutritive elements, into energy, and within certain limits store it up. In proper course this energy is transformed into work. Two intrinsic conditions may affect prejudicially the quality of the work done :—increased resistance to liberation of energy present in normal amount, and diminished amount of energy, the resistance to discharge being unaltered. In relation to the phenomena under consideration, the first of these conditions might result from inhibition, acting on the respiratory centres in such a way as to interfere with their normal discharging potential. It is quite conceivable that such inhibition would be effective if it directly influenced the respiratory centres in the medulla. Physiological experiments show that electrical, mechanical, and chemical stimuli directly applied to the medulla are capable of causing inhibition of the respiratory centres. Some recent experiments by Unverricht<sup>1</sup> tend to demonstrate the presence in the cortex of filaments (an actual inhibitory centre is not claimed) which, when stimulated, exercise an inhibitory influence over the respiratory centre. By means of a carefully graduated faradic current applied to a limited area of the cortex, Unverricht prolonged the duration of the expiratory pause. How inhibition can be produced by pathological changes, however, is not clear. If by inhibition we mean arrest of function in a centre, produced by a restraining influence extrinsic to it, the influence thus exercised acts in antagonism to the centre restrained. When effective, this implies greater energising power on the part of the controlling mechanism than on the part of that which is controlled. It is difficult to account for such a dominating influence being developed in any part of the higher cerebral tracts—its assumed locality—when they, as well as the respiratory centres, are labouring under the same defective nutrition. In the causation of Cheyne-Stokes breathing inhibition would need to be rhythmic ; if it were constant another factor would be necessary to account for the alternate gradual

<sup>1</sup> *Verhandlungen des Congresses für innere Medicin*, 1888.

rise and fall in the respiratory movements, either the immediate effect of changes in the chemical condition of the blood, or periodic overflows of energy from the cells inhibited. The former agency has already been discussed; the latter is a more complicated way of expressing fluctuations of energy at a higher level, which are about to be discussed under simpler conditions. The hypothesis of rhythmic inhibition would only remove causation a step back; the periodic nature of the phenomena would remain unsolved. In addition to difficulties in the way of accounting for the mode of production of inhibition, there are doubts as to its competence to produce the phenomena observed in the above reported case. It is questionable how far the respiratory centre, when under the influence of inhibition, can resist the powerful stimulus of excessively venous blood. In relation to this subject some of Head's<sup>1</sup> experiments are extremely suggestive. In producing apnœa by artificial respiration, Head found that as far as the advent of apnœa is concerned, it is immaterial whether the inflations are made with air, oxygen, or hydrogen, there is no observable difference. But there is a marked difference as to the several durations of the apnœa thus produced. With hydrogen the apnœa pause is short, and often the rhythmic breathing begins again immediately after cessation of the artificial ventilation. With air the pause is longer, and with oxygen the longest of all. The apnœa is not due to excess of oxygen in the blood, but to summation in the respiratory centre of the stimuli, caused by the artificially-produced periodic alterations in the volume of the lungs—a form of inhibition. The apnœa is sooner or later cut short by the advent of dyspnœa, which overcomes the after reaction of the inflations, and determines the return of rhythmic breathing. The interesting point is:—that the dynamic effect of the three gases is the same; the number of inflations required to produce the apnœa is identical in all cases. Starting with the same degree of inhibitory influence, we find that the rapid occurrence of dyspnœa, when hydrogen is used as inflating medium, quickly breaks down the inhibition and determines rhythmic respiration; whereas when

<sup>1</sup> *Journal of Physiology*, Vol. x.

oxygen is used the advent of dyspnoea is postponed, and the inhibitory influence exercises for a time undisputed control. I do not think in my case that, when the cyanosis was present, any stimulus could have determined an inhibition sufficiently dominant as to have rendered the respiratory centre insensitive to the exceedingly venous condition of the blood then existing.

The second of the two conditions postulated as influencing the quality of the work done by the respiratory centres—diminished amount of energy—remains to be considered. This hypothesis is in accord with the observations of most of the physiologists whose experiments have been mentioned and certainly affords the most consistent explanation of the phenomena which occurred in the case I have recorded. The progress of the case during life and the appearances after death, indicated clearly that interference with the blood supply to the central nervous system was the cause of the train of symptoms which occurred. The arteries supplying the brain and medulla were the seat of degenerative changes which had been in progress for many years. As the arteries became gradually obstructed, the nutrition of the dependent nerve-cells was progressively lowered, and they underwent slowly advancing molecular changes. The psychical powers were the first to succumb; the mental capacity, originally of the highest type, gradually faded. It is to be observed that the alteration was purely quantitative—simply a progressive diminution in intellectual energy without aberration. The first indication was, that problems which the patient formerly would have quickly solved on the spot, had to be reserved for deliberation. After a time the mind was unable to grasp the premises of an argument involving much co-ordinative power, and eventually it failed to comprehend isolated facts. Before this last stage was reached the physical powers were involved. Eventually the cell-groups concerned in the innervation of the respiratory apparatus suffered from the effects of the slowly increasing mal-nutrition, the result being a diminution in their energising power. As the action of the respiratory centre is probably automatic, apart from modification by external influences, voluntary and reflex, it would seem (in

response to some stimulus with the nature of which we are at present unacquainted) to be capable of liberating rhythmic impulses, each impulse being independent of the immediately antecedent condition of the blood, being simply the equation of the molecular activity by which the nerve-cells are energised. Under normal conditions there is probably stored up in the nerve-cells a reserve of potential energy that places the respiratory movements out of the reach of immediate want, so that a temporary slight derangement of nutrition is not attended by alteration of function. Serious disturbance of metabolism, however, resulting either from prolonged malnutrition, or from some interference of recent origin, lowers the activity of the cells, so that they are unable to maintain any reserve of energy; production and consumption then go on side by side. A very slight further reduction places the balance to the wrong side of the account, and continuous rhythmic respiration can no longer be maintained. As the available energy sinks, so do the respiratory impulses until the pause is reached. For a time there is no output, and during the early part of the pause the cells are probably quiescent. They then gradually resume activity until the energy developed reaches a certain potential, when discharge of impulses re-commences. This initiates the shallow breathing. The activity of the nerve-cells is still increasing and continues to do so for some time after the discharging point is reached, and as a consequence the impulses become stronger and the respiratory movements gain in amplitude. In the case narrated, when excess of bronchial mucus was present, there was, as stated, distinct cyanosis with pronounced air-hunger during the respiratory phases. At these times the undoubted excess of carbon dioxide and absence of oxygen in the blood, entirely failed to stimulate the respiratory centres into activity during the pauses; but on each occasion when breathing was re-established, the stimulus brought to bear by this condition of the blood exercised a powerful influence on them. This influence however, did not produce dyspnœal respiration immediately after re-commencement of breathing; the step by step development in amplitude of movements took

place as usual. These two facts have an important bearing as regards the interpretation just given of the mechanism of Cheyne-Stokes breathing. First, during the pause the excessively venous blood was unable to stimulate the respiratory centres into activity; second, when breathing had re-commenced it was equally unable to produce dyspnœal breathing until the centres had regained their full activity. It is evident that the same cause was at work in both instances. The hypothesis advanced assumes the cause to be exhaustion of the energising centres—complete in the first instance, and partial in the second. The absence of response was not due to blunted sensitiveness to stimulation, but to a more radical defect—want of energy, the cells were run down. Whilst the impulses increased step by step, the energy thus liberated was all that was being developed at the moment and it consequently could not be augmented by a stimulus. Stimulation can only liberate energy if it is present, or, if not potentially pre-existent, by developing the energising capacity of the cells concerned in its production. During the ascending group of respiratory movements the cells were doing the utmost permitted by their then state of activity—stimulation could not make them do more; but when their energising capacity was again in full swing, stimulation could, and did cause increased liberation of impulses, manifested by excessive amplitude in the respiratory movements, which continued until the first of the descending group leading down to the next pause, not as under ordinary circumstances, being limited to the first three or four of the fully developed movements. The natural result was more rapid exhaustion of the energising cells than usual; the periods of breathing being shorter under these conditions than when the bronchial secretion was in normal amount. It is possible that summation of stimuli in the respiratory centre, conveyed by the vagi, resulting from the excessive amplitude in lung movements, might aid in determining the earlier arrival of the pause. The three or four respirations of excessive amplitude, described as occurring after each pause under ordinary conditions, when the patient was free from the bronchial trouble, were probably, in some measure due to

slight excess in venosity of the blood, the balance being speedily re-adjusted.

The absence of distress during the pauses requires explanation. It may be accounted for either by supposing that the upper brain-tracts participated in the periodic exhaustion, or that exhaustion of the respiratory centres alone was sufficient to abolish for a time the sensation of air-hunger. The permanence of the psychical and to a lesser extent of the motor symptoms, indicates that the upper brain-tracts had undergone some amount of actual structural change. As regards the hemiplegia it is to be observed that although no changes were found in the hemisphere, the degeneration in the pyramidal tract points to antecedent destruction of some of the cortical cells or of their filaments. In addition to this, malnutrition would lower the activity of the structurally unchanged cortical cells and they would consequently readily succumb to exhaustion. That a condition of partial exhaustion of the sensorium periodically occurred is probable, but it is doubtful if it was to a degree sufficient to render the patient insentient to the dyspnœa. When the bronchial secretion was in excess the absolute indifference of the patient (not due to general insensibility) to conditions which only a few seconds previously had evoked the most acute manifestations of distress, indicated complete interruption of the stimuli which provoked them. I am therefore disposed to think that periodic exhaustion of the respiratory centres may have been the cause of abolition of the perception of dyspnœa. Hein,<sup>1</sup> Sacchi,<sup>2</sup> Ewald,<sup>3</sup> and other observers have noticed a condition of drowsiness during the pause, and in some of these cases the patients were sufficiently conscious during the respiratory phases as to explain that they felt no discomfort during the pauses, but that they suffered greatly from dyspnœa with each return of respiration, the distress beginning with the first shallow breath. In my patient there was no actual drowsiness, merely a state of passivity, with absence of perception of dyspnœa. His mental state and the aphasia, prevented

<sup>1</sup> *Deutsch. Arch. f. klin., Med.*, 1880.

<sup>2</sup> *Rivista clinica di Bologna*, 1877.

<sup>3</sup> *Berl. klin., Wochenschr.*, 1874.

any verbal expression of feeling, but his actions left no room for doubt as to what he did, and what he did not feel. It was most remarkable to see the patient in the midst of anxious manifestations of urgent dyspnœa, on some occasions of almost asphyxial violence, gradually tone down to a condition of perfect placidity with each successive step towards the pause. The return of the perception of the dyspnœa was simply an inversion of its loss. The first manifestation was synchronous with the first shallow breath, as the breathing increased in depth, so did the manifestations of distress.

The changes alluded to in the nerve-cells as occasioned by defective nutrition are probably of a purely molecular character, producing no alteration in the physical appearance of the cells, at least none that we can recognise. It is doubtful whether the microscopic changes found in the medulla bore any direct relation to the Cheyne-Stokes breathing. Influences of a much more subtle nature are at work, which, leaving the cellular framework unchanged, affect only the chemical processes taking place within the cell by which the oxygen of the blood is utilized as a mode of motion, so that the physiological function of the cell is interfered with apart from its existence as an anatomical unit. Imperfect metabolism would lead to this, both by diminishing the supply of oxygen to the cells, and by defective removal of effete products from them. In this way the normal activity of the chemical processes going on in the cells would be damped, and the sum of the resultant energy would be proportionally lessened. It is competent for results of this kind to follow quite recent blood-changes, whether quantitative or qualitative, and also after prolonged existence to quickly give place to renewal of normal function. In my case on three occasions at many months interval, the periodic breathing was replaced for some hours together by normal breathing. On one of these occasions there were alternations of periodic and regular breathing of several hours' duration, in accordance as to whether the patient was asleep or awake. West<sup>1</sup> relates a case in which Cheyne-Stokes breathing, unaccompanied by changes in the pulse or in the pupils during the

<sup>1</sup> *The Lancet*, 1890, p. 545.



pauses, extended over a period of three months. During that time it was replaced on one occasion by simple increase and decrease in the amplitude of the respirations without any pause, and on another by intermissions of two to four respirations, which replaced the pauses. These alternations are most easily accounted for by the hypothesis of exhaustion of the respiratory centres. The difference between fully developed Cheyne-Stokes breathing with pauses, and mere ebbing and flowing of the respiratory movements without pause, is due to the respective states of activity of the nerve-cells. In both instances their energising capacity was sub-normal; in one it was so much so as to necessitate a period of rest for recuperation; in the other, though below what was necessary to maintain regular respiration, it was sufficient to avert an actual standstill. A similar condition, it will be remembered, occurred in the early stage of my own case. The intermissions in West's case would be most likely due to slight depression in the energising capacity of the respiratory centres, with probably some altered sensitiveness to stimuli. The marvel is not that Cheyne-Stokes breathing sometimes undergoes modification in character, but rather that its phases are so regular in their sequence. This is the more remarkable when we consider that the abnormality of function is not dependent upon structural modification of the nerve elements concerned, but upon an altered state of their nutrition. The exceptional duration of my case makes it an extremely interesting example of perverted function without correlative tissue-change, apart from that affecting the vascular supply. The uncertain basis on which the Cheyne-Stokes breathing rested was evinced by the sudden reversions to normal respiration which occurred at protracted intervals, without any ascertainable cause, but presumably from a temporary improvement in the nutrition of the respiratory centres.

A feature of extreme interest in West's case is that when the patient during a pause was asked to take a breath, he only succeeded in setting in action some of the extra-respiratory muscles—the diaphragm and intercostals remained motionless. This limits the enervation to the ordinary



(automatic) respiratory muscles, and indicates that volitional impulses tending to modify the respiratory movements act on, or through, the respiratory centre as co-ordinating centre. Admitting the hypothesis of exhaustion, it is easy to conceive that the volitional impulses were arrested at the dormant respiratory centre (probably in the medulla) and consequently failed to reach the spinal motor nerve-roots.

## TREATMENT BY SUSPENSION.

BY J. S. R. RUSSELL, M.B., AND JAMES TAYLOR, M.A., M.B.

IN 1883, Dr. Motschoutkowski, of Odessa, published in the *Wratsch* a paper in which he detailed the results of treatment by suspension of several cases of disease of the nervous system—results sufficiently startling, especially as regards the cases of *tabes dorsalis*, to have merited something more than the scant notice they received at the time. The treatment, however, does not seem to have been practised by anyone until it was brought under Charcot's notice by a medical man, who, during a visit to Russia, had seen it carried out there. In the end of 1888, Charcot gave the treatment a trial, and in the beginning of last year, at one of his *cliniques* at the Salpêtrière, detailed his results. Although those were by no means to be compared with what the Russian physician obtained, still, backed by the great name of Charcot, they were such as to attract universal attention, and to ensure for the treatment a thorough trial. We have now had the experience of a year, and it may be well to review the results, in order to see whether the early promise of the treatment has been realized.

As to the manner of carrying out the treatment, Charcot gave directions—and all who have since practised it seem to have had these for their guide—that it was to be done with an ordinary Sayre's apparatus, using both head and arm pieces. The time allowed for the first suspension was to be half a minute, and each subsequent suspension was to be increased by the same period until a maximum of four minutes was reached. A direction was also given that the patient was to raise his arm every fifteen or twenty seconds, in order that the weight of the trunk might be thrown more and more on the neck alone. Suspension was to be carried out on alter-

nate days, and in thirty suspensions all the good likely to result was supposed to have been obtained.

Motschoutkowski, on the other hand, using the same apparatus and the same method generally, had suspended patients for a much longer time. Ten to twelve minutes was not an unusual duration, and five minutes seem to have been about the minimum after the treatment was fairly commenced. The course, too, was a much longer one, 97, 80, and 50 being some of the figures met with in his cases. Emphysema, phthisis, degenerated arteries, were to be regarded as contra-indications, and cases in which laryngeal crises occurred were regarded as unsuitable.

Motschoutkowski's list, as given in a translation of his paper in a recent number of *BRAIN* (Oct., 1889), comprised in all nineteen cases, twelve being cases of tabes, the others including cases of lateral sclerosis, chronic myelitis, disseminated sclerosis, and sciatica. There was no improvement except in tabetics. Of the twelve all improved except two, and those were early cases. The improvement consisted for the most part in disappearance of the paræsthesiæ and in diminution of the ataxy, but in three of the cases the improvement was remarkable. In the first there had been weakness and muscular wasting, reduced faradaic irritability of muscles, analgesia in the feet, ataxic gait, and unsteadiness in standing. After ninety-seven suspensions there was marked improvement — disappearance of paræsthesiæ, increased muscular force, and less ataxy. The second case was similar, but the change even more remarkable. He was confined to bed and unable to walk. After seven suspensions he was able to get up, after eleven he began to walk, and the improvement continued to advance as the suspensions went on. In a third case there was great weakness, muscular wasting, reaction of degeneration in the muscles, no impaired sensibility, but excessive pain. The pupil reflex was normal. After a long course of suspension, the muscles had regained their contractility, the pains had disappeared, he was able to walk, and the recovery was both complete and permanent. Such a case as this—especially as the patient was a publican—naturally suggests the picture of

multiple neuritis, and without the slightest attempt to question Motschoutkowski's diagnosis, it is well to bear in mind that such cases sometimes deceive the most experienced observers.

In none of the cases was there a return of knee-jerk, or any change in the visual apparatus.

None of Charcot's cases seem to have been so advanced as those just described. In March<sup>1</sup> he had treated in all one hundred and fourteen cases. In sixty-four the treatment was not sufficiently prolonged. Of the remaining fifty, thirty-eight improved, the gait was less ataxic—especially after each suspension, but after eight or ten continuously so—there was greater steadiness in standing, the bladder troubles improved, and so did the pains. There was no change in the condition of the reflexes. In one case out of three of Friedreich's disease, some improvement is said to have taken place. One case of disseminated sclerosis became worse; two cases of sexual failure improved. In eight cases of spastic paraplegia rigidity is said to have become less. Four cases of paralysis agitans are said to have felt better, and to have slept better; but no change occurred in the tremor.

Such results as those were quite sufficient to force this method of treatment under the notice of physicians, and we find lists of cases with detailed results scattered through the periodical literature of the last year. We propose now to refer to those results so far as they have come under our notice.

Morton<sup>2</sup> gives details of six cases of tabes. His best case shewed great improvement in walking, almost total cessation of lightning pains, return of pupil re-action to light, and return of sexual power. Optic atrophy, which was present, became no worse. There was similar if not so marked improvement in the other cases. Five minutes was the time allowed for suspension, and if the arms were not affected, the patient was allowed to suspend himself.

In the same journal Dana gives his experience of the

<sup>1</sup> *Progrès Médical*, June 22nd, 1889.

<sup>2</sup> *New York Medical Record*, April 13th, 1889.

treatment in six cases of tabes. He noted improvement in two—one after sixteen suspensions—the other after ten, no improvement in the others. Two cases of paralysis agitans are said to have felt better, nothing is said as to any change in the tremor. His general impression of suspension is that “it is a measure of minor and occasional value,” and after making this statement, he somewhat inconsequently goes on to suggest suspension by the feet, instead of in the orthodox manner.

Simpson<sup>1</sup> reports two cases. One was confined to bed but after a course of suspensions was “able to walk,” but the pains were still present: the other experienced improvement in pains and subjective feelings.

Watzfelder<sup>2</sup> has five cases of tabes. Those were suspended at first thrice daily for three minutes at a time, owing to a misunderstanding of the method. After five days of this somewhat heroic treatment, they were reduced to the ordinary alternate day treatment. They are said all to have shewn improvement—the gait becoming less ataxic, the incontinence less marked, and the pains less severe, although the improvement in the last respect was not constant. In one case “diplopia almost gone” is noted.

Eulenberg and Mendel<sup>3</sup> suspended thirty-four cases. In three the improvement was marked—consisting in greater control over bladder, diminution of pains, improved gait and increased steadiness in standing. One shewed a decrease in pains and paræsthesia alone. In none of the three noted as markedly improved, is there complete recovery from any one of those symptoms. In the majority of the remaining cases some improvement was present, affecting bladder function, gait, power of standing steadily, or pains, in four there was no improvement, one improved at first, relapsed later, and one was worse, having distinctly greater trouble with his bladder.

Ladame<sup>4</sup> relates his experience of fifteen cases of tabes, and one of Friedreich's disease. In two there was no im-

<sup>1</sup> *Canadian Practitioner*, June 1st, 1889.

<sup>2</sup> *New York Medical Record*, May, 1889.

<sup>3</sup> *Neurologisches Centralblatt*, June 1st, 1889.

<sup>4</sup> *Revue Médicale de la Suisse Romande*, June 20th, 1889.

provement; in the others there was slight improvement, similar to that experienced in the last series of cases. In two cases there was a return of the power of erection. No complete recovery of control over bladder is claimed. In his experience there was first a rapid improvement followed by a relapse, this being succeeded by a period of gradual improvement attaining its maximum after twenty suspensions.

Balaban<sup>1</sup> working under Dujardin-Beaumetz suspended nine cases. In one there was disappearance of pains, in four they were diminished, in two they disappeared but returned, in one they were not affected. The gait improved in eight, in the other a temporary improvement was succeeded by a relapse. In the same patient diplopia became worse; in one vision is said to have improved.

Haushalter and Adam<sup>2</sup> treated a number of cases suffering from the most varied nervous diseases including tabes, myelitis, neuralgic pains, hypochondriasis, &c., but as we have not yet sufficient data to discuss the effect in such diverse cases, we shall content ourselves with looking at their results in cases of tabes and spastic paraplegia. Of the latter condition they give two cases. No benefit was experienced. Of tabes they report six cases—two, early cases with pains, did not improve. The other four improved, two of them very distinctly as regards gait, pains, and steadiness; in one gastric crises persisted.

Bianchetti<sup>3</sup> details eight cases. In three the treatment could not be continued on account of faintness and amaurosis; in four of the remaining cases the results are described as wonderful, lightning pains, gastric and vesical crises, disappearing as if by magic. He produced no effect in some cases of paralysis agitans and impotence.

Marina<sup>4</sup> suspended twenty-one cases of tabes. In one-half there was marked relief, the remaining half became worse. In one case, a return of the knee-jerk—a violent jerk after five or six taps—is claimed to have occurred.

<sup>1</sup> *These de Paris, Progrès Médicale*, Nov., 1889.

<sup>2</sup> *Progrès Médicale*, Nov. 2nd, 1889.

<sup>3</sup> Quoted in *British Medical Journal*, 1889, vol. ii., p. 938.

<sup>4</sup> Quoted in *British Medical Journal*, *loc. cit.*

Michell Clarke<sup>1</sup> gives the results which he obtained in fourteen cases—twelve of tabes, one of Friedreich's disease, and one of doubtful nature. He got improvement in ten out of the twelve. The two who did not improve had ocular symptoms. The case of Friedreich's disease experienced no permanent benefit. Briefly, his conclusions are that this treatment increases the power of walking and standing. The pains usually become less after fifteen suspensions; relief of gastric crises is less certain, while knee-jerk, Argyll-Robertson pupils, vision and ocular paralysis are unaltered. It is to be noted that some of this observer's patients received a very long course of treatment, sixty, seventy, and eighty being some of the numbers met with, yet it does not appear that the results were much better than those met with in cases where thirty, twenty, or even twelve were the numbers.

Darier<sup>2</sup> suspended four cases in which optic atrophy was the leading symptom, and in all of them he reports improvement in vision.

Grainger Stewart<sup>3</sup> suspended five cases—three of tabes, two of spastic paraplegia. In all the improvement was marked. The pains disappeared, steadiness increased, and in one sexual power was regained. Two of the cases had only four suspensions, but the effect seems to have been equally great.

Bernhardt<sup>4</sup> suspended nineteen cases. He carried out the treatment every other day, and as he found only one patient who could bear suspension for four minutes, he made three minutes his maximum. In those cases there was diminution and occasional cessation of pains during the treatment; some are described as enthusiasts claiming wonderful improvement in walking while there was nothing objective corresponding to this fancy; one had erections return, with involuntary emissions so troublesome, that he begged the treatment might be discontinued. This observer evidently regards the treatment by suspension as being indebted for what good it does to the mental effect.

<sup>1</sup> *Practitioner*, Nov., 1889.

<sup>2</sup> *Recueil d'Ophthalmologie*, Nov. 4th, 1889, p. 249.

<sup>3</sup> *Edinburgh Medical Journal*, Jan., 1890.

<sup>4</sup> *Berlin Klin. Woch.* June 17th, 1889.

Erb<sup>1</sup> reports six cases. One became a little better, one at first better, later became worse, two were distinctly worse, and one died of an inter-current complication.

Dujardin Beaumetz<sup>2</sup> had twenty cases. In the majority at first some improvement, later a relapse to the former condition; in the minority no change. He is inclined to doubt the value of the treatment.

In the three cases recorded by Hale White,<sup>3</sup> one with pyrexia following suspension, the patients were apparently pleased with the treatment, although he notes that while in two the gait was rather better, and the ataxy less, in one of them pains and gastric crises became worse, and in the third, while the sensory symptoms were rather improved, the ataxy was worse and the other symptoms unaltered. One case had ten suspensions, treatment being stopped on account of the pyrexia. In the other two, the numbers were fifty and twenty-eight, while the time was gradually increased to seven minutes.

Dr. Althaus<sup>4</sup> mentions two cases of tabes, in one of which lightning pains ceased, while gastric crises did so in the other. In one case of paralysis agitans the tremor is said to have ceased for thirty-six hours after the first suspension. The same authority (*Brit. Med. Jour.*, Vol. ii., p. 872) reports a case in which ability to walk was restored, and the knee-jerk returned, but such a case is so remarkable as to make a much more detailed account of it desirable than is given simply in a letter to the Journal.

Dr. Saundby<sup>5</sup> has tried the treatment in six cases of tabes; and we cannot do better than quote his own words with reference to the effect of treatment. He says "It cannot be stated that any one of those cases derived benefit from suspension. One case certainly improved, but he was taking three-grain doses of iodide of potassium, three times daily, and he was also blistered down his spine."

Mr. Lum<sup>6</sup> reports five cases, one of whom improved

<sup>1</sup> *Neurolog. Centralblatt*, July 1st, 1869.

<sup>2</sup> *Therapeutic Gazette*, 1889.

<sup>3</sup> *Lancet*, Jan. 4th, 1890.

<sup>4</sup> *British Medical Journal*, 1889, Vol. i., p. 429.

<sup>5</sup> *British Medical Journal*, 1889, Vol. ii., p. 602.

<sup>6</sup> *British Medical Journal*, 1889, Vol. ii., p. 765.



considerably in walking power. He was taking iodide of potassium three times a day in fifteen-grain doses. The others remained *in statu quo*.

Churton, of Leeds,<sup>1</sup> suspended only one case, which became worse. He began with suspension for five minutes. In the communication in which he reports this case, he refers to another, which, while waiting for the suspension apparatus, improved so much as to be able to leave the hospital.

Such is the literature of suspension, so far as it has come under our notice. It will be observed that, although there is great variety both in the number and duration of the suspensions, the improvement claimed is fairly uniform. We have suspensions persisted in till eighty and ninety have been given, and improvement results; we have in some cases improvement almost equally well marked with three or four. So also as to the duration of the individual suspensions. While in some cases this is prolonged to ten or twelve minutes, in other patients it is found that three minutes is the longest that can be borne; while in still others three minutes three times a day is given, but whether it is given three times a day or three times a week, the results seem equally satisfactory.

We shall now proceed to lay before you the results of the suspension treatment as carried out by us at the National Hospital, Queen Square, under the direction of the physicians there. As a rule, no difficulty has been experienced in carrying it out. The majority of patients bear it well, and suffer little or no inconvenience; but the toleration varies considerably, and some patients always suffer more or less. In one of our cases nausea and vomiting, or a tendency to vomit occurred in one case during each of the four suspensions he had. Several had slight faintness with dimness of vision; a good many complained of some pain at the back of the neck, or in the jaws after suspension. Two had very severe pain at the lower part of the back at the conclusion of each suspension, and this remained present on each occasion. Other observers seem to have had similar experiences.

<sup>1</sup> *Brit. Med. Jour.*, 1889, Vol. ii., p. 818.

We have had, fortunately, no serious accident, but that the treatment is not without danger is evidenced by the fact that suspension has had already some victims. Two patients<sup>1</sup> were strangled from slipping of the chin-strap; a case is reported by Dr. E. Bloch<sup>2</sup> in which the patient suspending himself against the doctor's orders was killed; a similar accident occurred in a case reported by Dr. Gorecki. Another fatal accident was that which occurred to a medical man in America<sup>3</sup> suspending himself, apparently for experimental purposes. Dr. Borsari, of Modena,<sup>4</sup> reports a case in which serious symptoms came on in a patient after two or three suspensions of fifteen minutes each, and who died soon after of cerebro-spinal meningitis. It will be observed that in none of those cases were Charcot's directions closely followed.

In thirty-seven of our cases the treatment was carried out exactly as Charcot directed—viz., on alternate days, with a maximum of four minutes. In the other eight, suspension was carried out every day, with a maximum of three minutes' duration. The numbers of suspensions were as follows:—

12 had less than 20.

11 „ between 20 and 30.

22 „ over 30. Largest number was 50.

The cases were:—

Tabes...	...	...	...	...	...	32
Paralysis agitans	...	...	...	...	...	5
Ataxic paraplegia	...	...	...	...	...	2
Spastic	...	...	...	...	...	1
Functional	...	...	...	...	...	1
Neurasthenia	...	...	...	...	...	1
Doubtful nature	...	...	...	...	...	1

Of the forty-five cases nine improved; of these six were tabes, one was paralysis agitans, one was functional paraplegia, and one was of doubtful nature. Five became distinctly worse; the rest remained *in statu quo*.

<sup>1</sup> *Bulletin Médicale. Lancet*, p. 727., June, 1889.

<sup>2</sup> *Progrès Médicale*, 1889.

<sup>3</sup> *New York Med. Jour.*, June 11th, 1889.

<sup>4</sup> *Brit. Med. Jour.*, Vol. ii., 1889.

We shall now consider the cases which improved and those which became worse, with reference to the manner and degree of such improvement or deterioration.

Of the cases of *tabes* which improved, three were cases in which the improvement, although not great, was distinct and steady, consisting in a less ataxic gait and greater steadiness in standing. One remained subject to occasional lightning pains; another, although better, was still suffering from pain in the loins. Of the remaining three cases noted as improved, one was a very ataxic patient, who had first of all twenty-five suspensions without any change occurring. Soon after this, while having a course of baths in the hospital, he became much more ataxic, and suspension was resumed. After ten suspensions a steady and gradual improvement set in, and when he left hospital, after having in all forty-five suspensions, he was able to walk rather better than on admission. Considering the negative effect of the first course of suspensions, the improvement occurring during the second can scarcely be regarded as anything more than a coincidence. The other two cases of improvement experienced this to a considerable extent at first, having greater steadiness in standing and walking, but towards the end of the course of suspensions—in one of forty-one suspensions and in the other of thirty—they relapsed into their former condition.

So much for improvement occurring in cases of *tabes*. The other cases which improved were a case of paralysis agitans, a case of functional paraplegia, and one of doubtful nature. In the patient suffering from paralysis agitans, the improvement consisted of abolition of the tremor, which, however, he stated, occasionally returned at home after it had ceased to be present on the occasion of his visits to the hospital. The gait and the other conditions characteristic of the disease remained unchanged. The case of functional paraplegia improved a little, but as this is one of those cases in which one can confidently predict improvements with vigorous treatment of any kind, the fact that she did improve can scarcely be looked upon as shewing that there is any peculiar therapeutic value in suspension.

As to the case of doubtful nature which we have included among those who benefited by suspension, he came to hospital in August, complaining of pains in his legs, and after examination his knee-jerks were noted as absent. His pupils reacted normally, he had no inco-ordination, but slight starting of the tendons as he stood, no bladder trouble, no anæsthesia. Unfortunately his knee-jerks were not examined during the time he was suspended, but on examining him after his complete course—he was an out-patient—his knee-jerks were found active. His pains were less. What the nature of the case was it is not easy to say, but there is no evidence in favour of tabes except the absent knee-jerk, and as this was only observed on one occasion and by one observer, it can hardly be accepted as convincing.

Of the other cases, as we have said, five became worse while undergoing treatment, viz., three of tabes, one of spastic, and one of ataxic paraplegia. Of the three, one was a case pursuing a rapid downward course, and this course was in no way checked by suspension; a second was an extremely ataxic patient just able to walk with strong support, with bad cystitis and incontinence, and subject to pains and gastric crises. His treatment suffered interruption by a severe attack of gastric and vesical pain, accompanied with vomiting, and after it was resumed and completed, his cystitis and incontinence were still bad, although his bladder had been washed out daily; he was still subject to attacks of pain and his power of walking was lessened. The other case of tabes which became worse was an out-patient. After nine suspensions he remained away for a week on account of severe lumbar pain, and on returning his walking was manifestly worse. He had one more suspension, and then had an attack of erysipelas of the face, which did not allow resumption of the treatment.

The case of spastic paraplegia which became worse, was a young man with the disease affecting chiefly the left leg, and after he had had thirty suspensions this leg was considerably more rigid than it had been. The patient with ataxic paraplegia suffered from nausea or vomiting at or after each suspension, and although he had only had four he was weaker

and more shaky than he had been before the treatment was commenced.

Of the cases which remained *in statu quo*, most had ataxy, a few had occasional attacks of pains, some had cystitis and incontinence, one or two had gastric crises. Those symptoms underwent no change. Six had optic atrophy as a complication, of whom two were completely blind. No improvement was experienced in those cases, and in the cases where vision was less affected, careful testing could reveal no improvement, although one patient expressed the belief that he could see better. Dr. James Anderson has had this patient under observation since, and he informs us that there is now a distinctly greater impairment of vision.

It is evident that our results are not to be compared with those obtained by the majority of observers. While there are a few isolated observers, such as Bernhardt, Erb, Dujardin-Beaumetz, and Saundby, whose results are no less discouraging than ours, the great majority seem to have been far more fortunate. In the preceding lists there are in all 255 cases of tabes, of whom

171 improved,  
77 did not improve,  
7 became worse.

Our list included 32 cases of tabes, of whom

6 improved,  
23 did not improve,  
3 became worse.

To put it differently:—the percentage of improvement in other cases is 67 per cent., while in ours it is 18·7 per cent.; the percentage without improvement in those is 30·2 per cent., while in ours it is 71·9 per cent.; while in those cases 2·8 per cent. became worse, in our cases 9·4 per cent.

As perhaps explaining in part this great divergence, it must be remembered that while with any new treatment such as this, everyone who gets good results will at once be inclined to publish them, those who are not so fortunate and who are perhaps less sanguine, will not have the same eagerness to announce their unfavourable results. Two instances

in which this has been the case have come under our own notice, and we have no reason to suppose that our experience is unique. Then as to any great innovation in therapeutics we may be sure that its first year is its best. The enthusiasm for it dies down later, and the results are no longer seen in the rosy light which accompanies the dawn of any new treatment. We have only to look back a few years for an example of this, to the introduction of nerve-stretching for the relief of this same disease. The feeling with which a perusal of the results of that treatment in its first year will inspire anyone now is one of surprise, that any treatment offering such marvellous results should have fallen so soon into disrepute and disuse.

We have not yet cases of disease other than tabes in sufficient number to allow us to draw any definite conclusions as to the effect of suspension. Some cases of paralysis agitans have improved a little while being suspended, the majority have not. The results in spastic paraplegia are varied, while in disseminated sclerosis the general consensus of opinion is against any good effect of suspension. That the sexual apparatus is affected in some way in a certain number of cases is evident. That actual increase in power is caused is, we should think, extremely doubtful. Over excitation is a much more likely result, and whether this is to be regarded as an improvement or the reverse will depend on the nature of the case.

With the experience which we have had of suspension in cases of tabes it will readily be understood that we are not inclined to give the treatment much credit for relief, and far less for cure of any of the serious symptoms. In endeavouring to gauge the value of any therapeutic agent in this disease the peculiar and varying character of its manifestations must be borne in mind. One of our greatest authorities on nervous disease states the percentage of cases shewing no progressive tendency so high as 50 per cent., and every one knows that in many cases actual and marked improvement takes place both under treatment and independently of it. There was lately in this hospital, under Dr. Buzzard's care, a patient with tabes—a patient who, eight years ago, was

unable to walk from extreme ataxy and weakness, but who, during the last three or four years had been steadily improving and who is now (although the disease is well marked) able to walk about without even a stick, and to carry out efficiently the duties of military librarian in one of our garrison towns.

Reference has already been made to Dr. Churton's case, which improved distinctly while waiting for the suspension apparatus to be mended. Had this case been suspended, the improvement would naturally have been ascribed to the suspension, and such cases show how extremely careful one must be about *post* and *propter*. That ataxy varies at different times is well known. The familiar case of a patient who immediately after a severe attack of lightning pains has a marked increase of inco-ordination, illustrates this. Suppose such a patient towards the conclusion of the attack of pains subjected to suspension treatment, there is every chance that the cessation of the pains and the subsequent improvement in gait will be credited to the treatment, although numerous cases make it evident that improvement would follow even if nothing were done.

Lightning pains themselves come and go in the most unexpected way. Between one paroxysm and another there may be an interval of a few days; between that and the next there may be as many months; or they may vanish and never return. The very cases in which they have persisted for some time are those most likely soon to recover from them, for after all, their duration is limited, and this must be borne in mind in estimating the value of suspension, which by many is claimed as so efficacious in those pains. In none of our cases were pains a severe symptom before treatment. They did occur occasionally and they did not seem to be affected by suspension.

Gastric crises, too, relief from which, though to a much less extent than from pains, is claimed as a result of suspension, are just as variable and capricious as the pains. They, too, may vanish and never return, or they may return in a day or a year. That they had gone permanently could only be claimed after prolonged observation, and even then without a very large number of cases and without much



more uniform results than have as yet been obtained, their disappearance could not be ascribed to the treatment.

In none of our cases were gastric crises a very severe symptom. They had recently been present in very few, but where this was the case they continued to obtrude themselves at times. In one case a severe attack of gastric and vesical pain, accompanied by sickness and vomiting, necessitated an interruption of the treatment for a fortnight. We do not think that we should be justified in ascribing this crisis to the treatment, any more than we think others justified who do not hesitate to ascribe the absence of such symptoms to suspension.

The bladder troubles also in tabes are variable, and depend so much on mental conditions and on general physical conditions, both in and outside the body, that the greatest possible care is necessary in drawing conclusions as to the effect of treatment on them. In the majority of the cases reported, improvement in these symptoms is noted, but as in at least three cases—one of Michell Clarke's, one of Eulenburg and Mendel's, and one of Bernhardt's—there was greater trouble in this respect, while in another of Michell Clarke's, a tendency to incontinence was replaced by a tendency to retention, we must refuse to credit suspension with the improvement in one set of cases unless it is also made responsible for what happened in the other series. In our cases we have observed no change. Where cystitis was present the incontinence remained, and that too, although the bladders were daily washed out. In the other cases where urinary trouble was present, this consisted in slight difficulty in commencing the act, or in difficulty in resisting the call. In none of the cases was the symptom a troublesome one, and it remained unchanged. In the recorded cases in which incontinence was a symptom it is not stated whether cystitis was present and whether any local treatment was adopted. If the latter was the case, then it will become a question how much of the improvement is due to the local treatment. So far as we are aware, the only case in which a cure of cystitis by suspension alone is claimed, is one referred to at a recent meeting of the Medical Society of



London. Such a result is, of course, extremely unlikely, to say the least of it, and we should certainly hesitate to accept it without further details.

As to the increase or restoration of sexual power which is claimed as the result of this treatment, we have not observed it in any of our cases. A greater tendency to erection has manifested itself in at least one instance, but where sexual power was gone it has not been restored. Such a result of suspension, however, is claimed by some, while on the other hand one observer claims to have stopped the occurrence of troublesome erections by this treatment. In another case—one of Bernhardt's—there occurred during treatment an excessive tendency to erection with seminal emissions, so frequent and so distressing that the patient begged that suspension might be discontinued.

Passing by the question as to the advisability of restoring sexual appetite or function in tabetics, especially in cases where the disease is stationary, we can only say that this restoration after suspension treatment is by no means constant, and that instead of it the patient may experience a kind of false restoration, and may find himself in a condition much more distressing than his original state.

So much for the varying symptoms of tabes. But there are signs which do not vary, or at least not to the same extent, viz., ocular paralysis and impaired vision, the Argyll-Robertson pupil and the loss of knee-jerk. Of these there is perhaps the greatest chance of variation in ocular paralysis, especially in paralysis of the Levator palpebræ. In one case it is claimed that ptosis had disappeared, but as it frequently does so under the most diverse forms of treatment, its disappearance under suspension is not of much significance. In no case is it claimed that diplopia disappeared, but M. Darier claims improvement in vision in four cases with optic atrophy. Although a good many such cases are recorded, this observer is the only one who claims improvement. Among our cases are six with optic atrophy, and no improvement resulted in any, although one thought his sight better. Testing did not confirm this impression.

But more changeless than these are the loss of the knee-

jerk and, when it is present, the Argyll-Robertson pupil. In one case where the latter was present it is claimed that after suspension the pupil reacted sluggishly to light. Everyone knows how very difficult it is sometimes to be sure whether the reaction to light is present or absent. To illustrate this, we may mention a case which was seen about a year ago by a physician of eminence and in whom it was noted that the pupils did not react to light. Three months later the pupils reacted to light, and they do so still, and I think the physician himself would be the first to acknowledge that this was due, not to treatment, but to an error of observation in the first instance.

As to the return of the knee-jerk, the facts to establish such a change must be incontrovertible. The violent jerk, after five or six taps, claimed by Marina as a return of knee-jerk is to be regarded with the greatest suspicion. A very inactive knee-jerk cannot be elicited in that way, and we should think it is much more likely to have been a voluntary phenomenon. As to Dr. Althaus's case, in which the knee-jerk returned, as the case is only somewhat cursorily described in a letter to the *British Medical Journal*, we shall require further details before we can decide as to the real nature of the case. We think it may be laid down as an axiom that in a doubtful case the evidence of a solitary observer—no matter who he may be—as to the presence or absence of knee-jerk, should always be re-inforced by that of at least one other.

Such are the considerations which it seems to us necessary to urge before we arrive at a true appreciation of the value of any treatment of tabes. We have not referred to the mental effect likely to be produced by an impressive treatment upon patients who as a rule have tried many remedies, unfortunately in most cases with little success, and who are only too willing to clutch at even the shadow of a new means of cure. That such mental effect is considerable in some cases is certain, and how far it will account for that feeling of well-being, and of improvement in gait and balancing power, which wants the confirmation of any objective sign, it is not easy to say, but that some of the improvement is of this nature, I think, may be assumed. That it will

vary in different cases is evident, and we may possibly be able to detect national, as well as individual differences. It is not easy to decide how far such a consideration is to be allowed to influence a medical man in the treatment of a patient. That it should have some influence we think all will be prepared to admit, but to say as has been said<sup>1</sup> that "the patient is to be the final judge of treatment" is more than we should be prepared to admit. Whether this means that the patient is to have the last—and presumably the decisive voice—in deciding on the nature of treatment, or whether it means that the patient is to draw conclusions as to the value and efficacy of treatment in his own particular case—conclusions which are to be accepted by medical men—in neither case, we think, can the dictum be accepted. If it be accepted, the physician will certainly be relieved of considerable responsibility, but we think it will then be difficult for him to justify his existence at all. It seems to us that it rests with the medical profession to decide on the value of any treatment. It is for that body to examine and sift the evidence for and against; it is for the individual members to collect it, and lay it before that tribunal. We trust that our contribution may not be without its use in enabling an estimate to be formed of the value of the treatment by suspension.

<sup>1</sup> *Lancet*, Jan. 4th, 1890.

## PRELIMINARY NOTE ON CERTAIN MORBID PRODUCTS FOUND IN THE BRAINS OF PATIENTS DYING AFTER SEVERE HEAD INJURIES.<sup>1</sup>

BY ALEXANDER MILES, M.B., L.R.C.S., EDINBURGH.

WHILE engaged recently in investigating the microscopic appearances in a number of brains of patients who had died at varying periods, after receiving severe head injuries, I found in several cases large numbers of peculiar bodies, which appear to me to bear some relation to the so-called Colloid bodies described by various writers.

These bodies were found in the brain of a youth who died fourteen hours after sustaining severe injuries to his head by falling off the Forth Bridge; but as they were more characteristic in the case of a mason who survived his accident for two-hundred and fifty-six hours (256), I shall describe them from sections of his brain.

The bodies referred to vary in size from  $7\omega$  to  $50\omega$ ; the average being about  $18\omega$ . In shape they are more or less rounded or ovoid; are homogeneous, with no evidence of a nucleus, or other cellular contents, and except when very deeply stained are perfectly translucent. In some cases they exhibit a regular outline, the periphery being somewhat more deeply stained than the rest of the cell, while in others the margin is sinuous giving it the appearance of having shrivelled up slightly.

They take up the hæmatoxylin stain very readily, and stain black with osmic acid, but are unaffected by aniline-blue-black, and picro-carmin. The smaller examples stain faintly and uniformly, while the larger ones vary considerably in the degree to which they react to the dye. It would appear

<sup>1</sup> From investigations conducted in the Research Laboratory of the Royal College of Physicians, Edinburgh.

that these latter are formed by the coalescence of several of the former, as is shewn by their irregular outline at first, and later by their tense rounded appearance.

They are not confined to any one region of the brain, but are found in greater or less numbers alike in the cortex, basal ganglia, pons, medulla, and cord. To describe them as they occur in the cerebral hemisphere

They are distributed :—

(1) All through the white matter.

(2) In the most superficial area of the grey matter.

(3) In the “lymphatic system” of the brain.

(1) *In the white matter* they are exceedingly abundant, being thickly studded all through the section. Here they are fairly uniform in size and take up the stain well. They are largest, however, and most numerous in the immediate vicinity of punctiform hæmorrhages into the brain substance, where they not only invade the torn strands of nerve fibres, but also pass among the extravasated blood corpuscles.

(2) *In the grey matter* they are only to be detected in the most superficial layer, but there they are very plentiful. While they are less darkly stained and smaller than their fellows in the white matter, they shew a greater tendency to coalesce with one another, especially on the free surface of the brain, the largest being found in the epicerebral space.

(3) *In the “Lymphatic system” of the brain* they are chiefly found in one of two places, viz. :—

(a) In the meshes of the pia mater—the subarachnoid space.

(b) Along the course of the larger intra-cerebral blood vessels.

(a) It may be of importance from the point of view of their origin to observe that when these bodies are found in the *sub-arachnoid space* it is always in close proximity to a laceration of the brain substance, which makes it probable that they are then derived from the superficial layer of the grey matter. It is in this situation that they attain their greatest size, possibly because they have more room to expand here than elsewhere, or it may be that they swell up by imbibition of cerebro-spinal fluid.

(b) When in relation to *intra-cortical blood vessels* these bodies are found in the perivascular lymph space of His, between the hyaline membrane, or adventitia, and the brain tissue. They also are present for a short distance into the brain substance, and sometimes find their way into the space of Robin, between the tunica adventitia and the tunica muscularis, whether by natural or artificial openings is difficult to determine. The vessels round which they are seen are most evident in the superficial layers of grey matter, next in the region where the grey and white matters join—a very vascular area—and to a less degree as we pass deeper. In all respects they resemble those described in the sub-arachnoid space, save that they are somewhat smaller and more compressed laterally.

Of the exact significance of these bodies I am not at present prepared to speak definitely, but their general appearance, distribution, and reaction to staining agents, seem to point to their being in some way related to the myeline of the nerve fibres. Probably the main importance of the observation lies in the demonstration that these bodies may be produced very rapidly.

## Clinical Cases.

### COMPLETE PARALYSIS OF RIGHT THIRD NERVE IN A PATIENT AFFECTED WITH LEAD PALSY —RECOVERY UNDER THE DIRECT APPLICATION OF GALVANIC CURRENTS.

BY THOMAS BUZZARD, M.D.

*Physician to the National Hospital for the Paralysed and Epileptic.*

H. J., æt. 38, was admitted under my care into the National Hospital for the Paralysed and the Epileptic on February 4th, 1889, affected with complete paralysis of the right third nerve.

It appears, from notes taken by Dr. Taylor, Resident Medical Officer, that until five years ago his occupation had been that of a venetian blind maker, and that his health up to that time had been good. He then began to suffer from symptoms of lead colic, and a month or two later from "numbness," first in the little fingers and then in the other fingers of both hands. His hands became weak, so that he could not grasp anything strongly. He had also, he says, great difficulty in straightening his arms, and his hands hung from the wrists. His hands improved under treatment after about six months, but then he began to have attacks of headache, from which he has suffered from time to time ever since. The headache is described as affecting the right side of the head and forehead and being worse at night.

About a month after this headache commenced he had a fit, of which he knows nothing except that he bit his tongue severely. He had no return till eighteen months before admission into the hospital, when he was convulsed but did not bite his tongue. He had five or six of these apparently severe general fits, and then others of different and unilateral character. In these last he did not lose consciousness; the right side of the face was drawn up and the right arm and leg "worked up and down." Before the fit he always had trembling in the right arm and leg. In the attack he could not speak, though he understood what was said to him. He would have such attacks about every other day for eight or nine months, but none had occurred for four or five months before admission. He became an inmate of several hospitals in turn.

Four or five months before he came under my care he suffered from some loss of feeling on the right side of the head, but his

account of this is very indefinite. On January 31st, 1889, he woke up and found he could not open the right eye. There had not been any pain in the head just before.

The patient is married. His wife has twice miscarried, and he has three living children; the eldest delicate, very dull, and it would seem mentally affected; the others healthy. One died of some unknown cause. He denies any venereal infection.

On admission he was described as a pale, anæmic looking man, complaining of pains in the arms, weakness in the hands and legs. There was ptosis of the right eyelid, with abolished movement of upper, lower, and internal rectus muscles. The pupil was dilated, and did not react.

In the left eye Mr. Gunn reported that the edges of the disc were blurred, and there was a haziness over it.

A well-marked blue line was found on the gums.

In the arms there was but feeble power of extending the wrist joint, and the extensor communis digitorum and proprius pollicis failed to react to faradism. Omitting detail, the condition of the forearms was typically characteristic of lead paralysis.

The treatment adopted was at first indifferent, during which his symptoms remained unchanged. On February 13th he was ordered twenty grains, and on the 26th, thirty grains of iodide of potassium, to be taken three times a day. Under this the patient expressed himself as feeling generally better, and his arms stronger, but no difference shewed itself in the condition of the right eye.

On the 20th March, at his own request, he became an out-patient.

At this time he had been taking from sixty to ninety grains of iodide daily for thirty-five days, and there was no power whatever in lifting the lid or in moving either of the affected ocular muscles.

The constant current slowly interrupted was now applied in the following manner. A well-wetted plate rheophore was applied to patient's nape of neck, and connected with one pole of a Leclanché battery. The operator took the other rheophore well-wetted in his left hand, grasping the metallic portion in his palm, and applied the current to the patient's eye with the point of the index finger of his right hand. This was covered with one thickness of muslin, well wetted. The conjunctiva had been first rendered insensitve with a 2 per cent. solution of hydrochlorate of cocaine.

The strength of current which was employed marked from 1.5 to 2 m.a. on the galvanometer, and the alternate application



and lifting of the finger, making and breaking the circuit, gave rise to the feeling of an electric shock in the terminal joint of the operator's finger. The strength of the current was such as just to give rise to this sensation, and it was previously tested upon the patient's cheek.

Applications were then made in turn to the upper lid and the conjunctiva as near as could be contrived to the insertion of the internal, upper, and lower recti. The sitting occupied about two or three minutes.

A week later, on March 26th, when he was again seen, it was found that he could separate the lids by an eighth of an inch. The application was renewed, and on April 2nd it is noted that the "lids are now constantly apart. The pupils are equal. The right acts during convergence, but not for light."

On the 9th April Dr. Taylor's note runs thus: "The lower edge of the upper lid is now on a level with the upper border of the pupil. There is slight movement of the superior rectus—the inferior and internal recti remain as before. The pupils are now equal in size." He now felt his gait awkward, owing to erroneous projection, which he had hitherto been spared in consequence of the ptosis of the lid. There was marked diplopia. The galvanic applications were continued twice a week.

On April 16th it is remarked that the eye does not pass inwards beyond the vertical meridian of the palpebral aperture. On the 23rd the resident officer notes: "The right pupil is now slightly smaller than the left. Both react normally in accommodation. The right eye is as widely open as the left. The upward movement of the right globe is not quite so good as that of the left, and the downward is still more decidedly impaired, so also is the internal movement, but the eye now passes inwards beyond the vertical meridian of the aperture."

By May 15th the movement of the internal rectus was much improved, as likewise was that of the inferior, whilst the superior rectus appeared to act almost normally.

The patient was somewhat irregular in his attendance, so that the electrical applications were not made more than once a week.

In June the only defect remaining was an absence of reaction to light in the pupil of the right eye. All the external ocular muscles acted normally.

This case is marked by several features of interest. Although the gums presented a well-defined blue line, the patient had not been exposed to the action of lead in his

occupation for five years before he came under observation, and no source of more recent lead-poisoning could be discovered. There was a history of a few fits, apparently of epileptic character, the first about five years, and then none till eighteen months before admission, when he had a return of some half-dozen of the same character. These were followed by very numerous epileptiform attacks, apparently very different from the previous seizures, and pointing to some cortical lesion. The presence of optic neuritis lent confirmatory evidence in the same direction. The history of facial anæsthesia preceding the paralysis of the right third nerve is suggestive of some gross lesion in the basis cranii, affecting in succession the fifth and third nerves of that side.

What was the cause of these symptoms? It is natural, in the presence of a characteristic blue line, and of the typical features of lead palsy to ascribe the epileptic and epileptiform seizures, as well as the paralysis of the right motor oculi nerve, to the action of lead. But five years had past since the patient was, so far as is known, exposed to the action of that poison.

Moreover, although convulsive seizures are not unfrequently met with, I have never previously seen or met with any record of a case in which complete paralysis of a third nerve could be reasonably referred to the action of lead. It is true that Professor Westphal has recently published some cases of plumbic encephalopathy, in one of which paresis of one abducens oculi muscle was noted.

If the intracranial symptoms described were not due to lead it is, I suppose, most reasonable to refer them to syphilis, and to suppose that they were dependent upon gummatous deposit on the cortex cerebri, and also in the basis cranii. Such a concurrence of lesions is, of course, common enough in syphilis. To this view, however, there are two objections—one, not strong, that the patient denied any venereal disorder; the other, in my opinion a more weighty one, that although he was put under active specific treatment within fourteen days of the attack of ocular paralysis, no impression whatever was made upon this condition by large doses of iodide administered during thirty-five days. I could not

venture to say that this circumstance contra-indicated the syphilitic nature of the lesion, but I think that it throws some doubt upon it. On the other hand, the preceding lesion in the district of the fifth nerve, as well as the completeness of the eventual recovery, speak much more strongly in favour of syphilis than of lead, and appear to point pathologically to neuritis rather than to nuclear lesion.

The mode of application of the galvanic current in this case being unusual, and its results happy, I am tempted to give a few particulars of another recent instance in which the patient was favourably influenced by like treatment, though not to the same extent as in the case just related.

A married lady of about thirty-five years of age was seen by me in consultation on October 26th, 1886, suffering from complete paralysis of the left third nerve. For five weeks she had suffered from very violent pain over the left brow night and day, and for a week past this had been accompanied by severe and frequent sickness. Her condition shewed extreme suffering, and for several nights she had had no sleep at all.

She was ordered ten grains of iodide every four hours. On October 28th the pain was perhaps a little less, but still most severe. The dose was increased to fifteen grains. On the 29th she was still in great suffering. The iodide was continued, and in addition she was ordered calomel gr.  $\frac{1}{4}$ , with opium gr.  $\frac{1}{8}$  every three hours. She got relief after the first pill, and when I saw her on November 1st she was able to eat, and appeared to be greatly improved so far as the pain was concerned. The dose of iodide was now increased to twenty grains every four hours. On the afternoon of that day the pain returned with great severity, accompanied by retching and vomiting. Morphia was now given by injection.

On November 4th the pain quieted down, and was never afterwards severe. The condition of the eye, as regards the paralysis remained unchanged. There was at no time any conjunctival hyperæmia or tenderness of the globe. The ophthalmoscope shewed no change.

The mercurial treatment was continued to salivation, and the iodide was taken three times a day, until the end of November, without however producing the slightest perceptible effect upon the paralysis of eye muscles. Early in December she was seen by

Mr. Hutchinson, who was disposed to give an unfavourable prognosis. She continued during the month in the same state.

On January 12th, 1887, when all specific treatment had been suspended for some weeks I decided to apply electric currents. The left third nerve had then been completely paralysed for more than three months, and there was not the slightest sign of any disposition to return of power. On January 12th I applied a constant current slowly interrupted in the manner just described to patient's upper lid. After a few minutes' application there appeared to be a very slight return of power in the levator palpebræ. A few days later this was a little more marked. The application was now made to the various affected muscles, and there was immediate, though slight, improvement also in these, especially in the internal rectus. After four applications, the power of the levator palpebræ appeared to be perfectly restored, the left eye being opened as widely as the right. There was diplopia on looking to her right. At the same time on each occasion the power of the internal rectus was found to be increased, and after about eight or ten applications this muscle had entirely recovered.

The patient, it should be said, being professionally occupied, her attendance was very irregular, and sometimes at long intervals. In October, 1887, after about twelve applications I made the following note: "The left eyeball now rolls upwards better—the pupil is larger than that of the right side. It does not contract to light, but reacts slightly during the effort at accommodation. There is no action in the inferior rectus. The internal rectus acts perfectly."

In January, 1888, I found that when she looked to her *left* there was some diplopia, the images being described as about four inches apart. Examination now shewed that she failed to bring the cornea to the *outer* canthus.

In November last this inability had slightly increased so that the cornea, when she looked to the left, remained at about  $\frac{1}{4}$  inch. from the outer canthus. The diplopia necessarily continued. At that time there was some, but imperfect movement of the superior rectus and still less in the inferior rectus, though this was not absent. The internal rectus and levator palpebræ remained in perfect order. The condition has since not materially changed.

I would suggest the possibility that the apparent weakening of the external rectus may be due to some secondary contraction in the internal rectus muscle. One often sees in cases of peripheral paralysis of the portio dura that after recovery the contraction of previously paralysed muscles by over pulling the face gives rise to

apparent paralysis of the sound side, and it occurs to me that an analogous condition may possibly sometimes obtain as regards the internal and external rectus muscle of the eye.

We are justified, I think, in feeling that the striking and immediate effects which followed the application of the galvanic current in these cases is sufficient to indicate a relation of cause and effect, and not a mere association of coincidence. I have seen others of a similar kind, but my notes of them are not complete enough for publication.

As is well known the galvanic current is usually employed in such cases indirectly through the closed lids. It is hard to conceive that a current sufficient to influence the external ocular muscles can be carried to the requisite depth, through the resistance of the lids, without employing a strength which would be quite intolerable to the patient, and I have little doubt that the supposed galvanisation of these muscles through the lids is practically inert. The method of direct application described above was introduced by me in a paper published in the *Lancet* in 1875. It has been favourably noticed by Erb, but otherwise does not appear to have attracted attention. At the meeting of the Ophthalmological Society on July 5th, 1889, I demonstrated the mode of application.

## PARALYSIS ESPECIALLY OF ONE DELTOID MUSCLE IN A PATIENT SUFFERING FROM LEAD POISONING; PRESERVED ELECTRICAL REACTION OF THE MUSCLES.

BY THOMAS BUZZARD, M.D., F.R.C.P.

*Physician to the National Hospital for the Paralysed and the Epileptic.*

E. G., æt. 45, a house painter, was admitted into the National Hospital for the Paralysed and the Epileptic on October 8th, 1889, when the following notes were taken by Dr. Taylor, House Physician :—

Patient was in his usual health until three weeks ago, when he was suddenly seized with severe abdominal pain and constipation which yielded after three or four days to treatment. He then noticed that his hands and his shoulders, especially the left, were weak. For several weeks before the abdominal attack he had observed some weakness in both thumbs—a difficulty in abducting them, and a cramp-like pain in both little fingers. He had also noticed for some time past shakings in both hands, and this at times during the past summer had been so bad that he had been forced to stop work for a while. He had been obliged, for example, to employ both hands in taking a cup of tea. There had been, besides, pain in the left knee and both shoulders, and also in the left elbow. He had had no headache.

Five weeks before admission he had accidentally cut the back of his right hand with glass.

The patient had enjoyed good health, with the exception of occasional attacks of colic. In his work as a house-painter he had to do much “flatting.” He is married, and has six children all healthy, and his wife had had no miscarriages.

On admission, patient is a healthy-looking man, complaining of weakness in the hands and arms, especially on the left side, and pains in the knees. Nothing wrong is to be noted about the face and head. There is a well-marked blue line on both upper and lower gums. All movements of the fingers are carried out fairly well, but with tremor, and there is a tendency to flexion of the fingers at the metacarpo-phalangeal joints.

*Right Arm.*—The thumb is kept in a position of flexion at all the joints. Flexion, adduction, and opposition are carried out

fairly well, but with tremor; extension and abduction cannot be performed.

In the wrist the movements of flexion and extension as also ulnar and radial flexion and extension are carried out with fair power. Extension at the wrist is certainly the weakest movement. The movements of the elbow and shoulder-joints are good.

*Left Arm.*—There is here also a tendency to flexion at the metacarpo-phalangeal joints. The fingers however can be extended, and flexed, and separated from each other. The condition of the thumb is similar to that on the right side.

At the wrist all movements are carried out, but extension is executed feebly, and with tremor.

*Left Shoulder.*—The arm cannot be raised any distance from the side, nor can the extended arm be retained at a right angle to the trunk, or raised above the head. In attempts to do this much fibrillary contraction is noticed in the deltoid muscle.

Tremor is present when attempt is made to do anything, and this is equally well-marked in each hand. Fibrillary twitching is apparent in the forearms, especially on the extensor surface. There is no incoordination of movement. The elbow and wrist jerks are active and equal.

In the lower extremities all movements are carried out well, and there is no marked tremor. The knee-jerks are active and equal; there is no ankle-clonus; the plantar reflex is active and equal. Nothing wrong is to be noted in the thoracic and abdominal cavities. The urine is acid, sp. gr. 1020; no albumen; no sugar.

Examined electrically it is found that all muscles of both arms and hands—including the deltoids—react equally to a faradic current of a strength not greater, as far as can be judged, than would be necessary to cause contraction in healthy muscle. Reaction to the galvanic current is normal.

During patient's stay in hospital some little atrophy of the left deltoid became observable, but the electrical reaction continued normal.

Primary paralysis of a deltoid muscle in a case of lead poisoning, although rare, is by no means unexampled. Duchenne (de Boulogne), in his "Electrisation localisée," remarks: "The deltoid muscle is sometimes paralysed primarily," and refers to a case which he saw in hospital. The patient was affected with paralysis of the right deltoid consecutive (as in this patient) to several attacks of lead



colic. In Duchenne's patient, however, the middle fibres of the muscle had completely lost their faradic excitability, which still remained, but in a very feeble degree, in the anterior and posterior third. In the case which I bring forward the most remarkable feature is the preservation of electric excitability, of which I do not remember to have seen another example in a case of lead paralysis, where the loss of power was as complete as it was in this case. Indeed, the converse is what is to be expected. In an early number of this Journal<sup>1</sup> I pointed out that some muscles supposed by a patient suffering from lead paralysis to be healthy and unaffected, showed great diminution of faradic excitability, a point which had also been dwelt upon by Erb and Bernhardt before me.

When this case first came under my care a serious question arose in my mind (from observation of the electrical condition) as to the bona fides of the patient. But I soon came to a definite opinion that simulation was out of the question. The weakness of extensors shown by the tendency to flexion of the fingers and wrist, the tremor, the very distinct blue line, coupled with the history of repeated colic, bore powerful evidence as to the genuineness of the case. But most striking of all, perhaps, was the action of the man when asked to lift his elbow away from his side. Instead of alleging his incapability of doing so, he would make strenuous efforts to bring up his elbow, by employing the pectoralis major muscle in a manner unmistakeably characteristic of a case of deltoid paralysis.

Moreover, although on his admission no atrophy of the left deltoid was to be found, a perceptible thinning of its fibres occurred during his stay in hospital. Lastly, the man always endeavoured to make the best of his condition, and was with difficulty persuaded to remain in hospital, as he thought he could manage to do a little work.

When this patient was exhibited before the Neurological Society of London, at its meeting on November 21st, the preserved electrical reaction of the deltoid muscle was demonstrated, in my unavoidable absence, by Dr. Taylor.

<sup>1</sup> *Brain*, Vol. i., page 121.







ILLUSTRATIONS TO DR. SHARKEY'S CASE OF DIPHTHERIA,  
WITH ABSENCE OF KNEE JERKS.

## A CASE OF DIPHTHERIA, WITH ABSENCE OF KNEE JERKS.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

WM. C., æt. 4, was admitted into St. Thomas's Hospital, under my care on August 18th, 1888, and died on August 23rd, 1888. By some mistake I never saw the child during life.

He had been strong and healthy all his life (except for measles at eighteen months) until two months before admission, when his mother and her four children, the patient among the number, had sore throats, foul breath, and pain in the ears. No doctor was called in. After the throat got well the patient spoke with a nasal twang, fluids regurgitated through the nose, the voice got weak, so that he could barely make himself understood, and he became hardly able to walk.

On admission temperature was 98.6 F.; pulse 120, regular; tongue moist and coated with white fur; appetite said to be good, and bowels regular.

Body well nourished, no wasting of muscles observable. The patient used his arms and legs naturally, and could walk, but somewhat feebly.

Speech was indistinct and nasal in quality and difficult to understand. In drinking he choked a little, but there had been no regurgitation since admission. He made a snoring noise in respiration, and coughed a little. Soft palate and uvula did not move well. Throat not swollen or inflamed, but contained much mucus, chest moved well; no obstruction to the entry of air, physical signs normal. Heart normal. Knee-jerks absent. Urine 1025; no albumen or sugar. Eyes natural in every way.

During the rest of his life the temperature was often slightly elevated, and owing to difficulty in swallowing he had to be fed by a nasal tube. A trace of albumen appeared in the urine, and he got rapidly feeble.

On August 22nd he had two bad attacks of dyspnoea, attended with soft stridor. The diaphragm and inter-costal muscles were acting fairly well, and there was no evidence of pulmonary disease. As these attacks made feeding very difficult, and as the breathing was evidently obstructed in some way tracheotomy

was performed. On the day following it he had two or three attacks of dyspnoea, not so severe as before, and blueness and pallor came on suddenly. The pulse was fairly good and temperature normal. He died suddenly on August 23rd, apparently from stoppage of respiration.

*Post-mortem Examination.*—Body pale and thin, but well formed; heart, liver, lungs and spleen were all perfectly healthy in appearance. Lungs shewed here and there slight collapse, but were otherwise normal. Brain was generally hyperæmic, otherwise natural. Spinal cord looked healthy throughout.

The tonsils were somewhat enlarged, but free from membrane or any signs of acute inflammation. The soft palate and uvula were thick, almost cedematous in appearance. Larynx normal.

Thinking this was a favourable case for investigating the cause of the absence of the knee jerk in diphtheria, I hardened and had cut for me the spinal cord, the anterior crural nerve, and the quadriceps extensor muscle. The nerve and muscle proved to be perfectly healthy, but the spinal cord, though in the main normal, presented a very peculiar pathological alteration. The seat of it was in the large motor cells of the anterior columns; in them the change was very marked, but I was unable to make out any such alteration in the smaller cells of the posterior cornu. There were no evidences of inflammation to be found, and the other constituents of the cord were natural. The alteration in the multipolar cells is well reproduced in the figure, and consisted of a number of very large granules, completely filling the cells, and taking the logwood stain very deeply.

So numerous were they that they often completely obscured the nucleus. Their was a peculiar regularity about their arrangement, which is well seen in the figure, and the processes of the cells were often beset with them. In the latter situation they were long and appeared parallel with the direction of the process. The appearance was no ordinary granular condition, seen sometimes even in normal cords, but something quite unusual. Mr. Sherrington to whom I showed the sections said he had never seen such a condition before. On looking through a variety of preparations the only cases in which I found something of a similar kind were cases of myelitis.

The pneumogastric nerve I also examined microscopically and found it quite healthy. In several other cases of diphtheria which have come under my notice I have examined the paralysed nerves and muscles and found them healthy.

In the present case the alterations in function produced by

the disease were widespread and severe as was proved by the presence of the nasal twang and regurgitation of fluids through the nose: by the weakness of his voice, and his difficulty in walking: by the absence of knee jerks, difficulty in swallowing, the trace of albumen in the urine, and finally by his death from stoppage of respiration. I am lead to believe that the paralytic phenomena, and the absence of knee jerks were due in all probability to a change in the motor cells of the central nervous system, partly because the examination of nerves and muscles in this and in other cases proved negative, and partly because of the alteration found in the motor cells of the spinal cord.

The question naturally arises what gave rise to these changes. At present we are not in a position to make any definite assertion on this point. But if, as is probable, diphtheria is produced by micro-organisms, we can readily suppose that the vital changes which these organisms undergo in their growth upon the surfaces of the body and possibly in its interior produce certain poisons which being absorbed act specially as nerve poisons: and that the changes observed in the motor cells of the cord in the present case were the results of the action of such a poison. This is more likely than that the micro-organisms produce these results by their actual presence. For one cannot demonstrate their presence by the ordinary methods, and the paralytic phenomena often present themselves after all evidence of the original disease has disappeared, if we may judge by the absence of fever and of local inflammation. But the change in nerve cells produced by such a poison might well take considerable time to be recovered from. It does not seem to me that it would be fatal to this theory if in future examinations cases in which the knee jerk may have been absent, and the patient may have died from pulmonary or other troubles, no changes should be found in the cells of the spinal cord. For the poison might act in a lesser degree without producing serious disorganisation of nerve cells: and the knee jerks appear to be a singularly delicate test of the complete healthiness of the reflex arc in connection with them, and are abolished by slight causes.

If, however, changes should be absent in cases which have run so severe a course as the present, and which have been marked by serious and widespread paralytic phenomena, then the alterations found by me in the motor cells in this case will have to find some other explanation.

## Critical Digests.

### CHRONIC HYDROCEPHALUS.

BY M. ARMAND RUFFER, M.A., M.D.

(Continued from page 144.)

#### II.

OWING to the varying position of the lesions, it is impossible to give a clinical account applying to all cases of hydrocephalus.

The brain being distended by fluid, in one part or as a whole, it follows that the symptoms vary not only according to the amount of destruction of brain-tissue, but also according to the part of the brain which is the seat of disease. It will be better therefore to divide the subject into the same two groups, as in the part referring to the pathological anatomy of the disease, namely:—

1. Porocephalus, *i.e.*, Localised or total destruction of the brain tissue by a collection of fluid.

2. Hydrocephalus, *i.e.*, Distension of the brain by fluid without loss of continuity in the cortical substance.

*Porocephalus*.—The time at which porocephalus gives rise to symptoms is somewhat variable. In the majority of cases, however, the disease is present at birth or begins very early in extra-uterine life. Occasionally, the first symptoms appear after the patient has reached adult age. Unfortunately, in many of the published cases, the clinical description is very meagre. Taking into account, however, the cases only in which the beginning of the affection is noted with a fair amount of accuracy, it is found that in 55·5 per cent. the disease was congenital; in 6 per cent. the first symptoms appeared before the age of four months; in 75 per cent. before

the age of two years and in 86 per cent. before five years. In 5·5 per cent. the disease began between the ages of five and ten years, and in 5·5 per cent. of cases between the ages of ten and twenty. In 2·9 per cent. the patients were over twenty years of age, whilst, in one patient, the disease originated after he had attained the age of forty-five years. A distinction in that respect, however, must be drawn between the cases where the whole brain or a large portion of both hemispheres are absent, and those in which part of one, or a small part of both hemispheres are wanting. In the first class, the disease is almost always congenital, whilst the second class includes all the non-congenital, as well as some congenital cases.

*Total or almost complete absence of the Brain.*—In this first variety the symptoms call for little notice, especially as the children die at, or very soon after birth. A fact to be noted, is that the atrophy, or rather disappearance of the brain, has little or no effect on the development of the remainder of the body. These children are, as a rule remarkably well formed and sometimes, as in the cases recorded by Tarnier<sup>1</sup> and Budin,<sup>2</sup> very heavy. Convulsions are absent, and the patients cry, take the breast, pass urine and meconium just like ordinary children, though death, often supervenes very soon after birth. One patient whose case has been reported by Billard<sup>3</sup> lived three days, another (Tarnier) died after six and a half hours, another (Budin) lived forty-eight hours only, but Dickson<sup>4</sup> saw one who lived thirteen months. One of Rokitansky's cases (reported by Kundrat<sup>5</sup>) lived three months, the brain being wholly absent.

*Partial destruction of one hemisphere.*—The disease, when limited to one or part of one hemisphere, gives rise to symptoms, confirmatory of the knowledge as to the localisations of function in the brain obtained by modern experimental investigations.

Motor symptoms are often present, as the region of the brain which regulates movements is often attacked; the sylvian artery being very frequently the seat of morbid processes. Hence, hemiplegia on the side opposite to that of

the brain-affection is a common symptom, though, strangely enough, the paralysis does not often extend to the face. This hemiplegia is often complete, but, in some cases, one limb alone (arm or leg) is affected. In the majority of cases the paralysis is accompanied by atrophy of the limb and contractures, which almost always make their appearance at some time or other of the disease.

When the whole of one hemisphere is destroyed, the paralysis is, limited to the opposite side. This, however, is not always so, as in some cases, even when there is a unilateral lesion only, the paralysis is nevertheless bilateral. Cruveilhier,<sup>6</sup> for instance, in his atlas, gives an account of the *post-mortem* examination of a girl, who had been completely paralysed during life. After death the *right* side of the brain only was found to be converted into a cystic cavity. Epilepsy likewise, which is a rare (7 per cent.) symptom in this disease, affects both sides as a rule, even when the brain lesion is unilateral. When the lesion is limited to the motor region of one hemisphere, although the resulting paralysis and contracture affect one side of the body, the convulsions which occur in less than 10 per cent. of these cases only, are bilateral.

Among the most interesting cases described as having occurred in adults, is one described by Poncet.<sup>7</sup> A soldier who died of typhoid fever, a man of a somewhat low order of intellect, fell, twelve years before death, to the bottom of a well, and part of his brain was said to have escaped through the wound. He was *left-handed* but not really paralysed, and had never suffered from epileptiform attacks. The patient died of a fever, and at the *post-mortem* Poncet found a united fracture of the left frontal bone. There was a distinct loss of cerebral substance in the region corresponding to the middle of the first frontal convolution extending into the ventricle. The middle third of the second frontal convolution was atrophied.

Voisin<sup>8</sup> (Leçons Cliniques) relates the history of a girl who, at the age of thirteen, suffered from right facial paralysis, left spasmodic hemiplegia, atrophy of the upper limb and arrest of intellect. At the *post-mortem*, a cyst



was found in the situation of the right ascending frontal and parietal convolutions.

In cases where the anterior part of the brain alone is affected, motor symptoms are often almost absent. Thus, Jalan de la Croix<sup>9</sup> gives the case of a girl, seventeen years of age, who became slightly lame on the left side when two years old. At the *post-mortem*, the area supplied by the anterior cerebral artery was found converted into a cystic cavity.

Epileptic convulsions are not frequently met with and when present, affect both sides of the body, as a rule. Unfortunately, the character of the fits has not been accurately noted in many cases, and no mention is made as to whether the attacks begin in one limb, spreading to the other, or are bilateral from the first.

The limbs are often, though by no means always, in a state of contracture and this symptom may be often absent, even in well-marked cases of para—or hemiplegia. Sometimes, though rarely, the development of the limbs on the side opposite to the brain lesion becomes arrested.

The intellectual faculties are often of a low order, many of the patients being idiots, and Heschl<sup>10</sup> went so far as to say that porocephalus is always accompanied by idiocy. This is far too general a statement, as the disease is not incompatible with mental qualities of a high order. A girl, fourteen years old, for instance,<sup>11</sup> the bearer of a cyst occupying the left side of the brain was remarkably clever, earning her living as a sorceress, somnambulist and *physician* !!! Kundrat<sup>12</sup> showed that idiocy is frequently, though not constantly, found in congenital and occasionally in non-congenital cases, even when the first symptoms of the disease make their appearance in the second or third year of life. Hence, he argues that idiocy is not due to the lesion itself, but to faulty development and to atrophic processes taking place in one hemisphere. This is no doubt true for cases in which, the patient being an idiot, small lesions only are found in the brain, but where the whole of one or both hemispheres is destroyed it is easy to understand that the intellectual faculties in such a patient must necessarily

be of a very low order. Speech is often deficient, and the patient may even be completely aphasic. In most cases, however, the difficulty of speech is the result of idiocy caused by the disease. Binswanger<sup>13</sup> has lately published an interesting case in which the aphasia was clearly due to destruction of the speech-centre. A man who died when forty years old, had been born with an atrophied and paralysed right upper limb and a paralysed though not atrophied right lower limb. His intellect was not of a high order, and he had never been clever enough to go to school. Though not completely aphasic, the patient had marked defects in his speech. After death, the second frontal, the two inferior thirds of the ascending parietal, the first temporal convolutions together with the lobule of the insula were missing on the left side of the brain. The interest of the case lies in the fact that, although the speech centre had been destroyed on the left side (together with neighbouring convolutions), the centre on the other side had taken up its work, though rather imperfectly.

Nystagmus and deafness are occasional but rare symptoms. Sensory troubles are seldom met with, and the writer found them mentioned in three cases only, and even in these they were very slight. The symptoms therefore resemble those of spasmodic tabes and lobar sclerosis of the brain, a point which will be again referred to later on.

The course of the disease is generally from bad to worse, the fatal termination, in a large number of cases, being due to phthisis, or to persistent epileptic convolutions. Many (37·03 per cent.) die before the age of five years, 55·5 per cent. die before the age of twenty; 73·2 per cent. before that of forty, whilst a few live to the age of sixty, and three out of all the cases collected, lived more than sixty years.

*Hydrocephalus*.—Like porocephalus, the disease may be congenital or acquired.

Dr. Dickinson<sup>14</sup> found that out of twenty-six cases, four were congenital and sixteen began during the first six months, whilst six began at later periods up to the age of two years and two months. Dr. West<sup>15</sup> found that out of fifty-four cases, fifty showed symptoms before the age of six months, and that four manifested signs of it at birth.

Out of one hundred and three cases collected by the writer, nine were congenital, and in ninety-three patients the disease began before the patient was six months old. This does not represent the whole truth, for in many cases of foetal hydrocephalus the children are born dead, or destroyed during labour to save the mother, as the child is born alive in cases of slight hydrocephalus only. The manipulations to which the head is subjected during its extraction, and the faulty presentations which often occur, add to the sources of danger. Even after the body has been extracted, the bones of the head occasionally give way and extravasations of blood into the spinal canal, rupture of the neck also takes place.

The vast majority of cases of hydrocephalus, therefore, are congenital and if not actually present at birth, the disease generally makes its appearance within the first months of life. The head slowly enlarges, this increase in size being the first point to attract the parent's attention. Sometimes a fit, followed by contracture and paralysis is the first symptom or else the child cries out suddenly, is costive, restless, and shows all the signs of meningitis. Dr. West states that in twelve out of forty-five cases, fits were the first symptoms for some weeks. In six there was an attack like acute hydrocephalus and in four other instances the disease was preceded by some well-marked indications of cerebral disturbance. Squinting, nystagmus, headache are often early symptoms. As the head enlarges, the anterior fontanelle becomes more patent, the sutures widen and the head feels soft and fluctuating. In well-marked instances, the softness and thinness of the bones forming the cranial vault, the defects in the ossification, the presence of Wormian bones, together with the prominence of the fontanelles are quite characteristic. On holding a light on one side of the head, the cranium is sometimes found to be quite translucent. Fisher describes a peculiar soufflé heard over hydrocephalic heads. This sign is, however, absent in the majority of cases; and, when present, is useless for diagnostic purposes. The same may be said of *Cerebral Egophony*. It is reported that in one case a cracked pot sound, and a hollow sound in another could be obtained by

percussion of the patient's head. After death, a succussion splash on striking the patient's head has been noted.

The head enlarges slowly as a rule, but this increase is not always continuous. On the contrary, there is evidence to show that the disease may remain in *statu quo* for some time and then suddenly start afresh. A hydrocephalic child may be in fair health and suddenly be seized with a bilateral or unilateral fit caused by the increased pressure in the cranial cavity. The writer has seen a patient in whom the disease made no progress for ten years. Then, without any ascertainable cause, the child was seized with attacks of giddiness, restlessness and loss of control over both sphincters. In the case of a boy seven years of age, in whom the skull was already ossified, the head suddenly increased so much in size that bones were again separated. A protrusion of the brain may also suddenly take place externally, through a bone, or through a natural opening in the skull, as the anterior fontanelle may not close till the age of sixteen or even later. In fact, it appears that the clinical phenomena ought to be divided into two stages: the *acute* period in which the patient's head increases in size quickly, the symptoms being convulsions, squinting, fever, &c., and other signs of inflammatory mischief, and secondly, the *chronic* stage, in which the patient remains in fair health, the head enlarging slowly or not at all. Either stage may of course pass into the other.

Rupture of the brain is doubtless a very rare occurrence, and as far as the writer knows, there is only one case on record in which this is supposed to have taken place. Objections might, however, be brought forward even against that case.<sup>16</sup> That the skull occasionally gives way is fully established.

Brown<sup>17</sup> and Amyott<sup>18</sup> both published cases in which this had taken place. Leonard H. Sedgwick<sup>19</sup> gives an account of a hydrocephalic boy in whom the fluid was spontaneously evacuated through the nose. After the first flow of fluid the boy improved in health for some time, but became worse later on. Twelve months after it had ceased, the flow again took place, and when it ceased, the boy remained permanently cured.

In another patient, a bloody fluid oozed through the mouth and nostrils, and when the child died, an aperture was found between the cranium and nose through the ethmoid bone. Leber,<sup>20</sup> in 1883, carefully observed a hydrocephalic girl in whom a continuous flow of cerebro-spinal fluid took place through the nose; though, unfortunately, she was not cured by the discharge. This flow took place only when the patient bent her head forward, but this latter fact may be accounted for by supposing that when the head was in the normal position, the fluid passed through the posterior nares. Leber further suggests that some of the similar cases described by Nettleship, Priestley, Smith, Elliotson, Baxter and Sir James Paget, were cases of hydrocephalus. Dr. Sedgwick's case was cured, and Leber's was no worse for the accident; so that the conclusion to be drawn is, that this complication is not as fatal as might be expected, especially as we know that the fluid may escape without the brain actually giving way.

Fits, as a symptom of hydrocephalus, may be unilateral or, more frequently, bilateral, and are often followed by contractures and total or partial temporary paralysis. They almost always precede an aggravation of all the symptoms and are often the herald of a fatal issue. The upper limbs of a *still-born* hydrocephalic child,<sup>21</sup> have been found to be flexed and rigid, the fore-arms flexed on the arms, the hands on the fore-arms, and the fingers into the palms of the hands, the biceps-muscles being strongly retracted, and standing out prominently at the elbow joints.

Partial or general contractures are frequently met with in very young children, however, and like fits, generally mean a speedy death. Complete paralyzes are of very rare occurrence, and the patients, though often unable to stand, are usually, when in the recumbent position, able to move their limbs freely. Temporary hemiplegia, paraplegia even, have been noticed to follow convulsions. The eyes often look outwards and upwards, sometimes to the right and at other times to the left, varying in direction from day to day in the same patient. More rarely, they are directed downwards, or one eye only is affected. In one patient the

squint remained, even after the primary disease had been cured by appropriate treatment.

Bouchut<sup>22</sup> was one of the first to draw attention to the state of the *fundus oculi* in this disease. In 1865, speaking of the diagnosis between chronic hydrocephalus and rickets, he states that, as the first-named disease progresses, the following changes take place in the fundus :—

1. Great vascularisation of the papilla of the retina with dilatation of the veins, the latter, however, preserving their natural colour.
2. Augmentation of the veins of the retina.
3. Partial or complete serous infiltration of the papilla.
4. Atrophy of retina and vessels.
5. Almost complete atrophy of the optic nerve.

Similar changes have been described since, by Allbutt,<sup>23</sup> Swanzy,<sup>24</sup> Leber<sup>25</sup> and others; but these appearances are by no means always present in the *fundus oculi*, and there are but few cases on record in which such changes have been noticed. The blindness sometimes disappears when the child is cured of the primary disease. The writer has examined several patients without discovering any defects in their appreciation of colours, or in their field of vision.

Anosmia has been met with in some patients.

The state of the pulse and respiration does not seem to have attracted sufficient notice. In the cases the writer has seen, the respiration was normal in character and frequency, the pulse varying between 70 and 90, but in three cases the acceleration of pulse after the least excitement was much more marked than is normally the case, as the beats rapidly went up to 120 per minute and kept on at that rate for some time. In two out of fourteen patients, the writer found the patellar reflexes distinctly exaggerated. The other reflexes were normal.

The digestive system rarely gives rise to symptoms, though vomiting is sometimes one of the earliest signs which may persist through life. Costiveness has also been noted by several authors. Polyuria was noticed in one boy. Sleepiness and loss of control over the sphincters are occasional symptoms.

A young child suffering from hydrocephalus presents a characteristic appearance as a rule. When the patient is at rest in bed, the head, if very large, lies helplessly on the pillow and the sufferer is often quite unable to move it in any direction. The veins covering the head are enlarged, the skin is glazed and shiny, the cranium looking as if it was almost on the point of bursting, the scanty hair and the comparatively small triangular face together with the big head giving the patient an aged and worn appearance. The squint and perpetual nystagmus as well as the exophthalmos before noticed add to the strangeness of the child's appearance. In some cases, on pressure being applied to the anterior fontanelle, the patient falls into a kind of stupor which lasts as long as the pressure is maintained.

The child sometimes remains in one position for hours, but may be suddenly convulsed, sick, cry out or give other signs of agitation and pain. The limbs move about freely enough as a rule and sensation is normal, but the body, even if fairly large at birth, soon becomes puny and cachectic, especially should the head become much larger. Should the patient survive and reach the age of one or two years, he may learn to walk. The head is often too heavy for the child, and one hydrocephalic patient told the writer that the weight of his head often caused him to fall forwards. On examining hydrocephalic patients the writer has in three cases seen symmetrically placed on both frontal eminences a number of small scars evidently due to former falls. After a time, however, the patients may be able to walk well, but in slight cases of hydrocephalus even the body often remains undersized and weak. Thus an hydrocephalic girl, sixteen years old, only measured 74 centimeters in height.<sup>26</sup>

The intellectual faculties, even when the head is large, are often of a higher standard than would have been expected. Cardinal, the patient whose case has been placed on record by Bright, was fairly intelligent, and a patient whom the writer had an opportunity of seeing, earned his living by going about the streets singing comic songs and making stump speeches, by no means devoid of readiness and



wit. Bouchut<sup>27</sup> states that he knew two men above fifty, who, though markedly hydrocephalic, had intellectual talents of no mean order. The writer is acquainted with two similar cases. The first is a man fifty-three years old, and the other, a remarkably clever medical man, thirty years old. Others, however, even with smaller heads, are almost idiotic, for the state of the intellect does not altogether depend on the size of the head. Some never learn to read or write, appear to be insensible to kindness and often unable to speak. They cannot walk, look utterly idiotic, sit in one position with the saliva dribbling out of the half-open mouth, not taking any interest in things going on about them, hardly better, intellectually, than the lower animals. These cases are chiefly met with in lunatic asylums.

There is one fact which seems to have escaped the notice of writers on this subject, namely, the relation of hydrocephalus to deafmutism. In the writer's opinion the two conditions often go together. Through the kindness of M. Hugentobler, the writer was able to examine the fifty-one deaf and dumb mutes in his establishment at Villeurbanne, and found that thirteen of the inmates had very large heads, but that four of them, *i.e.*, about 8 per cent., were unmistakably hydrocephalic. These children varied in age from nine to twelve, were all fairly well-built, though three of them were distinctly undersized. They were quite deaf, not hearing the shrillest whistle close to their ears, but had all learnt to speak, though one of them had acquired the faculty of speech with a little more difficulty than the other pupils in the establishment. Physical examination of their eyes, ears and bodies, revealed nothing abnormal, except the large head above mentioned. In no case were there any signs of rickets, but one of them had syphilitic teeth and a suggestive scar in the back. One of these children was very clever, watching the speaker's lips with great attention and speaking French fluently and correctly. Two of them showed exaggeration of the patellar reflexes and the reaction of the pupils to light and accommodation was sluggish in all. Their digestive functions were normal, but M. Hugentobler informed the writer that some of them would go on eating



voraciously until told to leave off, though the same authority finds this to be frequently the case in deaf children. Their genital organs were well formed and none of them showed any inclination towards masturbation.

These children had never been ill and no cause, except hereditary syphilis in one case, could be discovered for their affliction.

Blindness and anosmia have been described, but the other cranial nerves are normal as a rule, though in one case a difficulty in swallowing liquids is noted.<sup>28</sup>

Unlike the majority of patients with congenital intellectual deficiency, the sexual appetite in hydrocephalic children appears to be normal, though in two cases an early precocity is noted in that respect.<sup>29</sup>

It is impossible to say what is the average duration of life in patients afflicted with hydrocephalus. With a very large head even (see case of Cardinal, reported by Bright), the patient may live to reach adult age, and the writer is acquainted with one man, aged thirty-nine, whose head is so large that he dare not show himself in the streets, who though undersized and puny, is in very good health and able to walk and talk. Bouchut knew of two cases above fifty years of age; and one seventy-one years of age, has been reported by Gölis.<sup>30</sup> When old these patients often become maniacal, melancholic or simply idiotic and end their days in asylums. The prognosis is, on the whole, very bad.

The cases recorded in medical literature, in which the patients have lived to adult age, are few, and the writer has been struck with the paucity of *post-mortem* records of adults. Nearly all such records refer to children under two years of age. In fact, on reading through the notes of the cases in medical literature, the author found that 68 per cent. died before the age of two years, and 80 per cent. before the age of ten years. Although these statements are, of course, only approximate, yet they give an idea of the duration of life in hydrocephalus. Death may be due to various causes, *e.g.*, repeated convulsions, marasmus, meningitis, or to intercurrent diseases, or to bursting of the head.

Dr. Blache stated that he had never seen a hydro-

cephalic child survive an attack of measles, and, as a rule, these children offer little resistance to intercurrent diseases. In some cases the head seems to have become less distended just before death and in Dr. Baxter's case the external protrusion through the frontal bone shrank visibly just before the fatal termination.

### III.

The conclusion arrived at was that in the large majority of cases of hydrocephalus the disease is congenital, though the influence of sex, heredity, &c., had not been mentioned.

Strangely enough, porocephalus is more frequently met with in females than males, whereas the reverse is the case in hydrocephalus. The latter fact has been noted by Morgagni.<sup>31</sup> Out of the cases collected by the writer: 59 per cent. of cases of porocephalus occurred in females, and 41 per cent. occurred in males. Whereas of hydrocephalic patients, 75 per cent. belonged to the male, and only 25 per cent. to the female sex.

Heredity has no influence in the production of porocephalus, as might well be expected, but it appears to play some part in the production of hydrocephalus. Out of twelve children<sup>32</sup> of one hydrocephalic patient, only one inherited his complaint, but one hydrocephalic woman<sup>33</sup> had four children all markedly afflicted with the same complaint. The father and brother of the hydrocephalic girl, whose case has been recorded by Leber,<sup>34</sup> had unusually large heads, but considering that only very few patients afflicted with this disease reach the age of maturity, it would be strange indeed if the affection was frequently hereditary.

The disease occasionally runs in families, or the same woman may have several hydrocephalic children in succession. In one instance, it is stated that, of two sisters, each gave birth to hydrocephalic children.

Dr. Hale White<sup>35</sup> gives the following account of the brothers and sisters of four hydrocephalic children:

1. Male child. Three hydrocephalic children are dead. One is hydrocephalic now.

2. Female child. Elder sister had an unusually large head.

3. Male child. Of eight brothers and sisters, one had an unusually large head.

4. Of four children from one mother, two have died of chronic hydrocephalus, one is now suffering from it, whilst one has escaped. The first three were born by instruments, but the last was born naturally.

G. de Grandmont<sup>36</sup> found that in one family two (three?) children out of six were hydrocephalic; and J. P. Frank records the case of a woman who gave birth in succession to seven children all afflicted with this disease. Similar cases have been noted by Underwood,<sup>37</sup> Armstrong,<sup>38</sup> and also by Simpson.<sup>39</sup> In Gölis's<sup>40</sup> work will be found the history of one woman who had six still-born hydrocephalic children. The seventh child lived eighteen months, and the two last were born healthy. According to Fritsch,<sup>41</sup> when several children of one mother are born with this affection, they mostly belong to the microcephalic variety. This is not the opinion the writer has formed from his reading, for he has been unable to find any family histories in which this had happened.

Syphilis has occasionally some influence in the production of the disease. In procephalus, the writer has found a history of syphilis in 4 per cent. of all cases only, but in so-called chronic hydrocephalus, he has found hereditary syphilis mentioned in 20 per cent. of the cases examined by him, or reported in medical literature. Poulet, however, gives the proportion as 10 per cent. only. Osiander,<sup>42</sup> Haase,<sup>43</sup> Rayer,<sup>44</sup> Gros,<sup>45</sup> de Méric,<sup>46</sup> Hutchinson,<sup>47</sup> Lancereaux,<sup>48</sup> Laschekewitsch<sup>49</sup> and quite recently again Sandoz,<sup>50</sup> have all drawn attention to syphilis as a cause of hydrocephalus. Haase<sup>51</sup> reports the case of a woman who had a chancre during the first half of her pregnancy, the husband being evidently syphilitic. She had three still-born children, but the fourth child was hydrocephalic, hemiplegic, with purple spots disseminated on the skin and died when six months old. The other children born in later years were evidently syphilitic.

De Méric<sup>52</sup> observed, on several occasions, an enlargement

of the head and peculiar rotatory movements in the eyes of syphilitic children.

Lancereaux<sup>53</sup> was told of a woman whose husband had contracted syphilis some years before marriage, and who gave birth to several hydrocephalic children; whilst Hutchinson<sup>54</sup> has noticed the co-existence of the two affections. According to Laschkewitsch,<sup>55</sup> syphilis may determine in the brain lesions of an inflammatory type, namely, inflammation of the ependyma, with consecutive inflammation of the ventricular liquid (?); this state being accompanied by other syphilitic lesions. This condition is characterised by continuous and progressive headache, nausea and vomiting, the symptoms being more marked when the patient moves about. The intellect becomes dulled, the pupils are dilated though contractile, and hyperæmia of the *fundus oculi* is noticeable with the ophthalmoscope. The pulse is unaltered, and there is no fever.

Georges Sandoz<sup>56</sup> believes in the existence of a syphilitic hydrocephalus, which may be congenital or develop in the first few months of extra uterine life. According to this author it is most probably caused by inflammation of the ventricular ependyma and plexuses, the morbid process being due to hereditary syphilis.

If syphilis be a cause of hydrocephalus it is difficult to understand why the latter disease is not more common. According to most authors, hydrocephalus occurs once in 3000 confinements, hence syphilis (syphilis being present in one out of five cases of hydrocephalus), acts as a cause of hydrocephalus about once in 15000 confinements. Now, syphilis is a common disease and it might be expected that, if it stood in any causal relation to the other disease, hydrocephalus would be more commonly met with. But, if in one out of five cases of hydrocephalus a distinct syphilitic history is found, it may fairly be argued that the first is sometimes the cause of the second, especially as to these cases might certainly be added some whose parents did not know they had suffered from that infectious disease, and some who wilfully conceal the fact of their having been thus afflicted.

In any case, syphilis is not the only cause of the disease, for the disease is described as occurring in animals (calves, goats, sheep, &c.), which are never affected by syphilis.

In 23 per cent. of all cases the writer found a history of tubercle in the families of hydrocephalic children, but never in the hydrocephalic fœtus, and in 4 per cent. only of the children who lived for some time after birth.

Poulet<sup>57</sup> states that hydrocephalus occurs chiefly among poor people, so that tubercle may possibly act as a cause, by weakening the health of the father and mother.

The coexistence of syphilis and tubercle in hydrocephalus, both being distinctly infective processes, raises the question as to whether, in some cases at least, the disease is not due to an infective process which ran its course in utero. We know now, that after the human body has successfully resisted an infective process, the evil effects of the latter do not always come to an end, even when the disease has to all appearances been mastered. During the course of scarlet fever, for instance, or after it has subsided, the urine may show that the kidneys have been affected by the infectious process, and though, in the majority of patients, the disease either ends in speedy death or perfect cure yet in some, long after all traces of the primary disease have disappeared, the morbid action set up in the kidneys still continues, may gradually add to the patient's danger, or even cause a fatal termination, although the real infective process came to an end long before the patient's death.

Syphilis is another example. A patient who has had syphilis, may become ataxic after all traces of the primary disease have left him. The sclerosis taking place in the posterior columns of his spinal cord is not a syphilitic process, for it resists the action of specific remedies, it differs from it in its anatomical characteristics and in its clinical course. Although the connection between syphilis and locomotor ataxy is, thanks to Dr. Gowers' work, established on a firm basis, locomotor ataxy, although often due to syphilis, is not a syphilitic process. Numerous examples (*e.g.*, in the ætiology of cerebro-spinal sclerosis, some forms of heart disease, &c., &c.) might be brought forward, to

show that an infectious disorder may set up after a time, a morbid process in some organs; this process, although not infective itself, being nevertheless due, in the first instance, to the primary infective disease. In the same manner, an infective process present in the father, mother, foetus, or newly-born child, might, even after the cure of the primary disease, possibly give rise to inflammation of the lining membrane of the ventricles of the foetus.

The frequent occurrence of hydramnios in the mothers of hydrocephalic children, together with the lesions found in the foetal vessels, also point to the disease being occasionally caused by syphilis.

Rickets coexisted with hydrocephalus in 13 per cent. of all cases, and has often been mentioned as a cause. Vinsoneau<sup>58</sup> especially lays great stress on the frequency with which the two diseases are combined. Considering, however, the wretched hygienic conditions in which most of these children are placed, the number of congenital cases and the frequent absence of all traces of rickets, the influence of the latter as a cause can only be very small.

Alcoholism, consanguineous marriages, advanced age of father and mother, epilepsy in the father, have occasionally been invoked as causes, but it is impossible to draw any certain conclusions as to their aetiological value. The forceps have been incriminated by some on the *post hoc ergo propter hoc* principle. The writer has been unable to find any confirmatory evidence on that point.

Dareste<sup>59</sup> has lately shown, that by subjecting eggs to somewhat rough usage, the embryo is often malformed in various ways. (Abnormal development of the brain, encephalocele, &c.) Blows, &c., to the mother might possibly cause the same in man, but the writer has only heard of two cases where this was supposed to have been the cause of hydrocephalus, and they neither of them bear close investigation. In 8 per cent. of all cases of porocephalus there was a distinct history of a blow, the onset of the disease being consecutive to the traumatism. Violence, therefore, must be admitted as a cause. In hydrocephalus, external violence has been reported in about 2 per cent. of cases only, but has been noted by as eminent an observer as Dr. West.

Cretinism, as a cause of hydrocephalus, has been mentioned in almost all text-books. The author cannot say why this idea should have gained ground, for he has been quite unable to find any confirmatory evidence. The only case in favour of that opinion is that published by Stoltz<sup>60</sup> in which a cretin gave birth to a hydrocephalic fœtus. Poulet made inquiries on the subject in Switzerland, and was unable to gain any confirmatory evidence as to any causal relations between rickets and hydrocephalus.

Syphilis, hereditary tubercle, blows and deficient hygiene, are therefore the only causes known to favour the development of the disease.

The ætiology of porocephalus calls for a few special remarks. Many causes have been invoked to account for origin of the porocephalus.<sup>61</sup>

Breschet<sup>62</sup> has attributed most cases to an arrest of development. In a great many *post-mortem* examinations, however, traces of old hæmorrhages, disease of vessels, &c., have been noted. In these cases arrest of development as a cause of the disease must be excluded. Moreover, the remainder of the brain presents, as a rule, no other malformations. In a few cases, however, the presence of hare-lip, fissure of the face, malformation of other convolutions seem to point to some early fault in development.

Encephalitis has been assumed by some to act as a cause, but it appears to the writer without there being any definite facts to support that theory. The pathological anatomy of the disease reveals the fact that the lesion is often exactly limited to a vascular area and changes (obliteration) of the vessels of that part, produced through thrombosis or embolism, are frequently found at the autopsy.

It would serve no useful purpose to discuss whether the changes in the vessels are primary phenomena, or secondary to changes in the placenta and heart, as Kundrat supposes. It is sufficient for our purpose to know that the appearances have been described by many, and among others by Duret, Budin, Klebs, Kundrat,<sup>63</sup> and that the cyst-like formation in the brain is sometimes due to disease of the vessels of the cortex.



Where the cortex is nearly intact, the cyst is often formed by the dilatation of one of the horns of one ventricle. The pathological cavity may communicate with the ventricle, or may be cut off from it by a band, the result of a previous inflammation. Blows seem to have been the primary cause of the disease in a few cases.

Porocephalus is therefore the result of two distinct processes :—

A.—Disease originating in cortex (blows, hæmorrhage, encephalitis, &c. A form generally accompanied by loss of cerebral substance).

B.—Disease originating in ventricles.

There are no real points of difference therefore between cases of porocephalus belonging to the latter category and hydrocephalus. The liquid accumulates in the brain in both, but whereas in the first it distends one ventricle or part of one ventricle only, in the latter it distends both ventricles though not always in an equal degree.

#### IV.

The brain of a patient suffering from hydrocephalus is distended and the cerebral substance compressed owing to the increased quantity of fluid present in the ventricle. As the accumulation of fluid increases, the pressure on the surface of the encephalon rises *pari passu*, owing to the cerebro-spinal fluid pressing on the lymphatics and on the walls of the veins of the arachnoid membrane.

It has been proved experimentally, that when the intracranial pressure is increased, the venous flow is arrested first; but that if the pressure rises still more, the flow in the capillaries comes to a standstill, and the anæmia of the brain substance thus produced gives rise to symptoms of compression of the brain.

Leyden<sup>64</sup> has proved by experiment, that an increased pressure of the cerebro-spinal fluid in animals gives rise to restlessness, groans, screams, irregular respiration, together with marked slowing of the pulse.

Epileptiform attacks with opisthotonos, extension of the



hinder extremities, deep and irregular respirations, followed by coma and flaccidity of the limbs, dilatation of the pupils, increase in the rapidity of the pulse (300 per minute or more), vomiting, loss of control over the sphincters follow in quick succession. A very gradual increase of pressure does not produce these effects.

Acute symptoms similar to those are present often enough in hydrocephalus, though they are sometimes absent from first to last; the intra-cranial pressure, owing to the yielding of the skull, being less than might be expected.

When convulsions and other symptoms occur, it may be assumed that they are due to a sudden augmentation of intra-cranial pressure. The presence of paralyses, contractions, &c., calls for no special explanation, and the occurrence of optic neuritis must be accounted for in the same manner as it is in other cases of increased intra-cranial pressure.

Most of the symptoms, as well as the disturbances in intellect, may possibly be caused or made permanent by the changes which, in one case, the writer has proved to have existed in the cortex of the brain.

The central canal having been found closed in a number of *post-mortem* examinations, it has been suggested by many that the distension of the skull and the symptoms of the disease were due, solely and simply, to the obstruction to the flow of the cerebro-spinal fluid and to the increased intracranial pressure produced by the slow and gradual distension of the cranial cavity. Many facts, however anatomical, clinical and pathological are opposed to this view.

The ventricular cavities are not closed sacks, communicating by one foramen only, with the cerebro-spinal canal and arachnoid cavity; for since the classical investigations of Key and Retzius and others, it is an admitted fact that there are many points of communication between the ventricles and the outer surface of the brain. The fourth ventricle communicates with the sub-arachnoid spaces, firstly, by the foramen of Magendie; secondly, by the foramen of Bochdalek and the *apertura ventriculi quarti*

*laterales*, first described by Luschka and rediscovered by Axel Key and Retzius.

The ventricles of the brain communicate with the fourth ventricle through the aqueduct of Sylvius, but Key and Retzius have found that, by injecting coloured substances under low pressure into the sub-arachnoid spaces of the spinal cord, a direct communication between them and the hemispheres of the brain could be traced in both animals and human beings; this coloured substance distributing itself into the velum interpositum, and also into the choroid plexus. The lateral ventricles always contained some amount of free fluid which may penetrate through the aqueduct of Sylvius into the fourth ventricle, and even into the central medullary canal. These communications certainly suffice for the evacuation of the fluid normally present, even after the closure of Magendie's foramen. But whether all these openings, including Magendie's, would be sufficient when, through inflammation, a large quantity of fluid is poured into the cerebro-spinal cavity is very doubtful; for we know that large serous membranes like the peritoneum, are often quite unable to absorb the inflammatory fluid poured into their cavities.

The pathological liquid found in the ventricles is not normal cerebro-spinal fluid, but differs from it in the fact that it contains far more albumen, and a larger quantity of certain salts, is of higher specific gravity, may be turbid and contain pus-corpuscles, is more highly-coloured and resembles in some respects the product of an inflammatory liquid.

Though an obstruction has sometimes been proved to exist, yet in many *post-mortem* records there is no mention of any impediment to the flow of liquid being discovered. Magendie<sup>65</sup> himself reported cases in which the foramen called after him was not only open, but was even larger than natural. In a remarkable case of very large hydrocephalus published by Gaucher,<sup>66</sup> this competent observer distinctly states that the fluid ran with the greatest ease through the foramen Magendie into the arachnoid cavity. In Mr. Hutchinson's<sup>67</sup> patient, the hydrocephalus communicated by

a large canal with the spinal bifida, in the lumbar region. It may also be mentioned that, in the horse, the foramen is normally closed, and that in man, Axel Key and Retzius have found it closed without its giving rise to hydrocephalus.

It is difficult to understand why, when there is no obstruction present, the fluid should not, following the laws of gravitation, descend into the spinal canal. The writer can offer no explanation of it, except by calling attention to the fact that, in other cases of accumulation of fluid in the head, the liquid does not always find its way into the spinal canal.

The progress of the disease is not always a gradual one, as it certainly would be supposing the clinical phenomena to be due merely to a slow and gradual accumulation of fluid, but the morbid process may remain quiescent for years, may stop at any time and may even be cured, if convulsions giddiness, and other symptoms do not point to the super-vention of further mischief in the cranial cavity. Thickening of the ependyma by interrupting the flow in the veins of Galen and by obstructing the central canal may possibly give rise to some retention of cerebro-spinal fluid, and this retained liquid may itself act as a further cause of irritation; the beneficial effects of tapping being explained by the removal of one source of irritation.

We know, for instance, that some forms of ascites are cured, or improved for a time, by a judicious tapping. If, however, the cause of disease is a chronic inflammation of the membrane lining the cavities of the ventricles, and if, as in most chronic processes, sudden exacerbations sometimes take place, then the character of the fluid, the occasional sudden onset of the disease, the convulsions, headache, vomiting &c. find a ready explanation. If due to mere continuous passive accumulation of fluid, it is difficult to understand why the disease should occasionally stop in its progress, or even be cured. If the cause is an inflammatory process (one resembling for instance, that giving rise to a hydrocele) it is easy to see that, when the morbid action has run its course, or when treatment has successfully interfered, the patient may remain in *statu quo* or permanently recover.

Tubercle and syphilis have already been alluded to as important ætiological factors. Now tubercular patients are, of course, subject to remissions and to exacerbations, but this is especially the case with syphilis. A syphilitic patient may remain in perfect health for some time, without showing any signs of the disease, but under the influence of bodily or mental ill-health, the syphilitic symptoms suddenly reappear. This is exactly what happens in hydrocephalus, and the writer assumes that, in some cases at least, the sudden exacerbations may be due to a new outbreak of the syphilitic virus.

The presence of fluid, and the symptoms, in the writer's opinion, are not due to mere passive accumulations of fluid in the ventricles, but are the result of an occasional acute, but usually chronic inflammation of the membrane lining the ventricle. The obstruction is secondary to the primary inflammation and although possibly intensifying, is not necessary to the production of the disease.

If hydrocephalus be compared with other congenital defects of the cerebro-spinal axis, several points of resemblance with diseases which, at first sight, appear to be very dissimilar, may be found. A true myelocoele, as Virchow has shown, is always due to the dilatation of the central canal of the spinal cord. If this dilatation be slight, the cord is almost normal in appearance; but should more liquid accumulate, the spinal cord ruptures, the bony walls of the spine atrophy through pressure and the tumour protrudes externally; that is, posteriorly, laterally, or even anteriorly. If, for the sake of simplicity, we assume the brain to be a tube surrounded by walls thicker than those of the spinal cord, the same process will produce similar effects, but the *post-mortem* appearances will vary according to the time at which the morbid action has begun. Let us suppose, that in early foetal life, whilst the skull is almost unossified, a ventricle or part of one ventricle slowly dilates. The cyst thus formed, whether it be cut off from the remainder of the ventricle or not, gradually increases in size and perforates the skull, not protruding through a fontanelle as a rule but through a bone, and an encephalocele is the result. In

another patient, the brain has been partly or almost completely destroyed through disease in early foetal life, the convolutions are absent, or only traces of them exist, the encephalon is represented merely by a large cyst, and the foetus presents the characteristics of hydrocephalus or porocephalus. In still another patient, this dilatation has begun late in foetal or early in independent life, *i.e.*, when the brain is already formed, and the bones developed to some extent.

The effusion of liquid has taken place into a closed cavity which expands by slow degrees, giving rise to a large hydrocephalus. Like a spinabifida and like an encephalocele, hydrocephalus may become latent, *i.e.*, not increase in size, or may suddenly enlarge, so much so that the walls may suddenly give way.

Spinabifida is frequently associated with hydrocephalus and hydro-encephalocele. In fact, in some patients in whom that disease begins in the neck, there may be a hernia of the brain and spinal cord through the *sanæ* foramen. In others (*e.g.*, in Mr. Hutchinson's) the whole cerebro-spinal system is affected. It is only fair to assume that the same pathological process which produces hydrocephalus in the brain, gives rise to a myelocele when it attacks the spinal cord or to both, when both the brain and spinal cord are affected.

The various dilatations, therefore, which occur in the cerebro-spinal central canal, the writer has arranged under the following heads:—

1. Dilatation of the central canal of the spinal cord giving rise to a myelocele. This may burst during foetal life and form a spinabifida occulta.

2. Dilatation of one ventricle or one horn of it, forming a porocephalus. This may protrude through the skull and is then called an encephalocele; or it may burst after working its way through the skull, and give rise in early foetal life to an encephalous monster.

3. Dilatation or destruction of both hemispheres in early foetal life, with premature ossification of the skull. (Porocephalus.)

4. Dilatation of both hemispheres late in foetal or early in independent life. (Chronic hydrocephalus.)

## V.

The difficulty in the diagnosis of hydrocephalus depends largely on the extent of the lesion and on the size of the patient's head. In cases where the whole head is enlarged to a slight extent only, the disease may be taken for one of hypertrophy of the brain, a mistake which, according to some authors, is by no means uncommon. It must be remembered, however, that the probabilities are always in favour of its being a case of hydrocephalus; and secondly, that symptoms such as paralysis, contractures, fits, optic neuritis, point in the same direction. Intellectual defects, moreover, are rare in hypertrophy of the encephalon, but frequently present in hydrocephalus. So little, however, is known of the former disease, that a slightly marked case would present the greatest difficulties.

The fontanelles and the whole cranium are enlarged in the rickety child, just as they are in hydrocephalus, but always to a far less extent. The presence of a well-marked chain of Wormian bones, the gaping of sutures are in favour of the disease being hydrocephalus. Lastly, the rickety deformities in other bones, with a head only slightly enlarged, point to the disease being due to rickets only.

In a well-marked case the diagnosis is easy, as there is no other disease in which the head attains the enormous dimensions which have been recorded. If the disease be limited to part of one hemisphere or to a few convolutions—when the head is not enlarged—the diagnosis becomes extremely difficult and it is almost impossible to form an opinion in such cases. The only difference between hydrocephalus and the disease due to chronic meningo-encephalitis, &c., is the absence of hemiplegia in the latter.

It differs from atrophic sclerosis of the brain, only in the fact that the latter is frequently not congenital, and more often secondary to infectious diseases. (Richardière).<sup>68</sup> Lastly it has to be distinguished from the diseases giving rise

to hemiplegia in children and especially from the one-sided paralysis due to vascular lesions. It is needless to enter into the diagnosis between it and cerebral tumour, or tubercular meningitis, the symptoms being entirely different.

Vascular lesions of the cerebrum are excessively rare in childhood, although hæmorrhages have been described by a few authors. The writer fails to see how they could be distinguished from porocephalus either in their mode of onset or clinical course. The same applies to the endarteritis due to syphilis, although the latter might possibly be distinguished through the effects of treatment. Lastly, we must not forget that vascular changes may themselves produce porocephalus.

In conclusion, therefore, although the diagnosis of the well-marked forms is quite easy, that of the slighter forms is in almost all cases almost impossible.

### TREATMENT.

Porocephalus admits of no treatment either preventive or otherwise, but the treatment of chronic bilateral hydrocephalus must be divided into two parts, namely, surgical and medical.

The surgical procedures which have been advocated are :—

1. Tapping.
2. Pressure and tapping combined.
3. Injection of iodine.
4. The introduction of a seton.
5. Permanent antiseptic drainage.

West, in 1845, collected all the cases treated by puncture (56 in number) and found that the cures amounted to a little over 28 per cent. By adding Dr. West's cases to those the author has collected (69), the number of successful cases after tapping amounts to 21·75 per cent. only. Hydrocephalus is an almost hopeless disease, however, and any procedure therefore which cures one in five cases and which, as a rule, does no harm (for in the writer's cases only three seem to have actually suffered from the effects of the puncture) may conscientiously be tried. König<sup>69</sup> advises the use of a trocar previously disinfected by heat and carbolic acid.



An assistant exerting some pressure with his hands on the head, the instrument is to be introduced at a spot about 265 centimetres distant from the sagittal suture and is to be pushed, avoiding the longitudinal sinuses, right into the ventricle. It is as well to draw the skin over the seat of puncture beforehand so as to form a valve-like opening. Thompson<sup>70</sup> drew off 10oz. and some more escaped afterwards. Bruns<sup>71</sup> advises to draw off 2-4oz. at a time, but should the fluid flow readily more may be drawn off with advantage. The ease with which the flow takes place may therefore serve to some extent as a guide to the amount of fluid which is to be drawn off.

Malgaigne<sup>72</sup> lays down the following rules:—

The operation may be performed,

1. In a patient three or four months old, even when the hydrocephalus appears not to be increasing.
2. In a patient over four months old, if the head be not already ossified, and if the hydrocephalus increases and constitutes a danger to the life of the individual.

Bruns advises the operation only if the distension is considerable, if the fontanelles and sutures are widely open, if the bones of the head are free and mobile, if the child is well nourished, not paralysed and is physically and intellectually as advanced as a normal child of the same age, and lastly if the hydrocephalus be continually increasing in size.

It is impossible to draw up a code of rules to be applied to all cases. Some cases get well spontaneously, so that if the disease is not actually progressing, it is only right to see what nature will do, but if, after using drugs, mercury, iodide of potassium, &c., &c., the child steadily gets worse, the head ought to be tapped. The presence of paralysis need not be a counter indication, for cases have been recorded in which the child, though blind and paralysed, recovered sight and power of walking after the cure of the primary disease.<sup>73</sup> If one tapping be not sufficient, the operation may be repeated successfully as in Thompson's case, though it is but seldom that, after one has failed, repeated operations have any good effect.

Compression has been warmly advocated by many,



especially at the beginning and the middle of this century ; but has deservedly fallen into disuse again. Rivierius in 1656 mentioned pressure as a cure for hydrocephalus. Sir Gilbert Blane in 1821 claimed to have cured hydrocephalus in that way, and in 1823 Barnard<sup>74</sup> speaks of six cures thus obtained in his own practice, and mentions that Dr. Engleman was successful in ten patients. Trousseau<sup>75</sup> for some time advocated pressure, but having seen a patient die in consequence of it, condemned the practice.

The development of the effusion, says Trousseau, being limited at its superior part of the skull owing to the compression<sup>76</sup> "the base had given way just as it gives way when, in order to separate the bones of the skull, anatomists fill it with water and beans which, swelling up, produce the disarticulation." Dr. Dickinson<sup>77</sup> lost one case through sloughing of the scalp during compression but Phillips<sup>78</sup> reports that (on a boy whose head was almost on the point of bursting) he tried the application of pressure ; the child recovering and being alive and well ten years afterwards. He mentions that the same method was also successful on another of his patients.

Injection of iodine has been tried several times, notably by Winn, Barnard<sup>79</sup> and Tournesko, but has as far as the writer is aware only been successful once in the case reported by Tournesko.

A seton has been used always with a fatal result. Haven<sup>80</sup> in one patient tried permanent antiseptic drainage but with a fatal result, and electricity is reported to have cured one patient.

Medically, hydriodate of potash and the iodides of potassium and iron have been used without success. Bromide of potassium has been advocated by Brunton<sup>81</sup> who has cured a case by its use.

If, as the writer supposes, the disease is frequently associated with the syphilitic diathesis the use of mercury is distinctly indicated and there is some evidence that the latter drug is useful, Gölis<sup>82</sup> and Gowers<sup>83</sup> have both cured cases in that way, and Umpleby<sup>84</sup> in the *British Medical Journal* of 1871 has recorded a remarkable case of cure by its use.

The child in question was twenty-two months old, had been blind for six months, and its head measured 27 inches in circumference. Dr. Umpleby ordered :—

R.

Hyd. c. Cret.	...	...	...	} aa	gr. ii.
Rhei.	...	...	...		
Sod. Carb. ...	...	...	...		

or,

R.

Hyd. c. Cret.	...	...	...	gr. ii.
Pulv. Ipec. c. Op....	...	...	...	gr. i.

according to the child's condition.

Three months afterwards the child was able to walk, had, at three years of age, recovered its sight perfectly, and was, except for its large head, quite well. Dr. Umpleby further states that his father cured a child in the same way. In a case of progressive hydrocephalus the writer thinks that mercurial treatment might be tried first with advantage, having recourse if it failed to tapping.

It is my pleasant duty to thank my friend, Prof. Pierret, for suggesting this subject to me, as a thesis, for much advice and for kindly allowing me to make use of the vast pathological material of Bron Asylum.

#### LIST OF REFERENCES.

- <sup>1</sup> Tarnier, *Loc. cit.*
- <sup>2</sup> Budin, *Loc. cit.*
- <sup>3</sup> Billard, *Traité des maladies des enfants nouveau nés*, 1828.
- <sup>4</sup> Dickson, *Lancet Loc. cit.*
- <sup>5</sup> Kundrat, *Loc. cit.*
- <sup>6</sup> Cruveilhier, *Loc. cit.*
- <sup>7</sup> Poncet. *Soc. de Biol. Paris*, 1880.
- <sup>8</sup> Voisin Leçons cliniques.
- <sup>9</sup> Jalan de la Croix, *Virch. Arch.* xcvii.
- <sup>10</sup> Heschl, *Praager Vierteljahresschrift*, 1859.
- <sup>11</sup> Lambl *Arch. f. Phys.* V. xii.
- <sup>12</sup> Kundrat, *Loc. cit.*
- <sup>13</sup> Bülowwanger, *Loc. cit.*
- <sup>14</sup> Dickinson, *Lancet*, 1870.
- <sup>15</sup> *West Med. Gaz.*, 1842.
- <sup>16</sup> See Bright, 'Med. Cases.'
- <sup>17</sup> Brown, *Med. Times & Gaz.* London, 1869.
- <sup>18</sup> Amyott, *Loc. cit.*
- <sup>19</sup> Sedgwick, *Med. Times & Gaz.* 1856.
- <sup>20</sup> Leber, *Arch. f. Ophthalm.* Berlin, 1883.
- <sup>21</sup> Golay, *Bull. de la Soc. Anat.*, 1873.

- <sup>22</sup> Bouchut, *Comp. Rend. Soc. Med.*, 1865. *Comp. also Paris Med.* 1884.
- <sup>23</sup> Allbutt, *Loc. cit.*
- <sup>24</sup> Swanzy, *Loc. cit.*
- <sup>25</sup> Leber, *Loc. cit.*
- <sup>26</sup> Fürst, *Arch. f. path. Anat. Berlin*, 1884.
- <sup>27</sup> Bouchut, *Loc. cit.*
- <sup>28</sup> Loir, *Revue Med.*, Nov., 1843.
- <sup>29</sup> Lyons, *Lancet*, 1830-31, p. 654. Forbes Winslow, *Ibid.*, p. 735.
- <sup>30</sup> Gölis, *Prakt. Behand.*, Vienna, 1818.
- <sup>31</sup> Itard is responsible for this statement.
- <sup>32</sup> Personal observation.
- <sup>33</sup> *Ib.*
- <sup>34</sup> Leber, *Loc. cit.*
- <sup>35</sup> Quoted by Dr. Baxter, *Loc. cit.*
- <sup>36</sup> G. D. Grandmont, *Bull. de la Soc. d'Anthropologie*, Paris, 1884.
- <sup>37</sup> Underwood, *Loc. cit.*
- <sup>38</sup> Armstrong, *Dubl. Quart. Jour. Med. Science*, 1848.
- <sup>39</sup> Simpson, 'Obst. Works,' London, 1856.
- <sup>40</sup> Gölis, *Loc. cit.*
- <sup>41</sup> Fritsch, Halle, 1881.
- <sup>42</sup> Osiander, quoted by Lancereaux.
- <sup>43</sup> Haase, *Ibid.*
- <sup>44</sup> Rayer, *Ibid.*
- <sup>45</sup> Gros, *Ibid.*
- <sup>46</sup> De Méric, *Lancet*, 1858.
- <sup>47</sup> Hutchinson, quoted by Lancereaux.
- <sup>48</sup> Lancereaux, *Traité d'Anat. Path. and Traité de la Syphilis*
- <sup>49</sup> Laschkewitsch, *Centr. of Chir.*, 1880.
- <sup>50</sup> Sandoz, See *Hayem Review*, 1887.
- <sup>51</sup> Haase, *Loc. cit.*
- <sup>52</sup> De Méric, *Loc. cit.*
- <sup>53</sup> Lancereaux, *Loc. cit.*
- <sup>54</sup> Hutchinson, See Lanceraux.
- <sup>55</sup> Laschkewitsch, *Loc. cit.*
- <sup>56</sup> Sandoz, *Loc. cit.*
- <sup>57</sup> Poulet, *Loc. cit.*
- <sup>58</sup> Vinsonneau, *Loc. cit.*
- <sup>59</sup> Dareste, *Gaz. Med. de Paris*, 1880.
- <sup>60</sup> Stoltz, *Mem. Med. Soc. Strasburg*, 1861.
- <sup>61</sup> Breschet *Bullet. Fac. de Méd. de Paris*, 1820-21.
- <sup>62</sup> *Arch. gén. de méd.* 1831.
- <sup>63</sup> See Chap. on pathological anatomy.
- <sup>64</sup> Leyden, quoted by König, *Allgem. Chir.*
- <sup>65</sup> Magendie, quoted by Axel Key and Retzius.
- <sup>66</sup> Gaucher, *Bull. Soc. Anat.*, 1879.
- <sup>67</sup> Hutchinson, *Trans. Path. Soc.*
- <sup>68</sup> Richardière, *Thèse de Paris*.
- <sup>69</sup> König, *Allgem. Chir.*
- <sup>70</sup> Thompson, *Med. Chir. Trans.* 1874.
- <sup>71</sup> Bruns, quoted by König, *loc. cit.*
- <sup>72</sup> Malgaigne, 'see Jaccoud's Dictionary'—Hydrocéphalie.
- <sup>73</sup> Umpleby, *Brit. Med. Journal*, 1871.
- <sup>74</sup> Barnard, *Lond. Med. Report*, 1825.
- <sup>75</sup> Trousseau, *Clin. de l'Hôtel Dieu*.
- <sup>76</sup> *Ibid.*
- <sup>77</sup> Dickinson, *Loc. cit.*
- <sup>78</sup> Phillips, *Lancet*, 1857.
- <sup>79</sup> Barnard, *Loc. cit.*
- <sup>80</sup> Haven, *Bost. Med. and Surg. Jour.*, 1882.
- <sup>81</sup> Brunton, *Glasgow Med. Jour.*, 1872.
- <sup>82</sup> Gölis, *Loc. cit.*
- <sup>83</sup> Gowers, quoted in *Reynold's Med.*
- <sup>84</sup> Umpleby, *Loc. cit.*

## Reviews and Notices of Books.

*A Text-book of Mental Diseases*: with special reference to the Pathological Aspects of Insanity. By BEVAN LEWIS, L.R.C.P.(Lond.) ; M.R.C.S.(Eng.) Charles Griffin & Co., London, 1889.

THIS is in many respects the most important book on Insanity extant. As one lays it down an inclination arises to compare it with the works of Bucknill and Tuke, Griesinger and Maudsley ; but reflection shows this to be impossible, as the condition of scientific knowledge of the anatomy, physiology, and pathology of the nervous system differed at the various periods at which each was written. Bevan Lewis has the advantage of being the youngest man on the list, and of having worked in an atmosphere cleared by his predecessors of much of the mists of tradition which hung around the subject. All the works cited must live as successive enunciations of the philosophical methods of the study of insanity, and each marks a mile on the road. Lewis has added another mile, or perhaps two. He has had the path greatly cleared for him, and has had brilliant light thrown on it, afforded by the results of recent investigations. But his work is by no means solely based on the observations of others ; he has worked with his own hands, and has taken a prominent place amongst the men who have done so much of late years to open up the secrets of the nervous system. His arduous labours have been productive of many important results, and it is curious to note how meagre has been the recognition of his work in Germany, and even in this country ; we know of no man who has done so much solid work, who can append to his name such a small list of honorary distinctions.

In one respect this book is unique :—it begins with an elaborate exposition of the anatomy of the brain and spinal cord. If we mistake not, this is the first treatise on insanity which deals in a thorough manner with the anatomy of the organ implicated. It may be thought by some, that for the practical purposes of the work the first six chapters are unnecessarily diffuse, and that a

more general description would have sufficed. But the author probably thought it well to provide the student of nervous disease with ready access to full information, holding that as such knowledge becomes more generally diffused so the probability of the explanation of obscure clinical phenomena will be increased. Lewis writes on Meynert's lines, and occasionally in Meynert's obscure style. Not having availed himself of the recent results of the developmental method of tracing fibre, certain of the descriptions of tracts in the medulla (*e.g.*, the fillet and olive), and of the relations of nerves to nuclei (notably the auditory) are not entirely satisfactory. The two chapters on the structure of the cerebral cortex, however, are in every respect admirable. A well-known investigator remarked to us lately that they contain the very best description of these regions in this or any other language, and this opinion we unhesitatingly endorse. They should be at the fingers' ends of every man who attempts to treat brain disease, for, without full and intimate knowledge of the anatomy of the convolutions, no one has a right to advance even a theory of the genesis and progress of pathological processes occurring in them. Whoever does so is in the position of the man who, unacquainted with the structure of the kidney, treats of "dropsy." This applies specially to the vascular and lymphatic systems of the cortex and adjacent white matter; and of both a clear and vivid description is afforded. The implication of these systems is at the root of most disturbances of consciousness, and the author cannot be too highly complimented on the manner in which he has demonstrated their arrangement. It is open to question whether he does not attach too much importance to the action of the "lymph connective" tissue cells. This, however, is a question which must remain *sub judice* for some time to come. And, again, it is fair to take exception to his statement that the channels between the brain substance and the hyaline sheath "are not the lymph spaces proper," but that they communicate with the epicerebral space of His. His holds that this is a lymph space, and it is, therefore, somewhat difficult to reconcile the two opinions, as it is not easy to realise the existence of non-lymphatic channels debouching into a lymphatic cavity. Our own opinion is strongly against the existence of any epicerebral lymph-space, and we hold that the channels are mere involutions of the naked surface of the brain.

This section concludes with an all too short and too restricted reference to physiology. In a work which deals so fully with

anatomy it might have been expected that the functions of tissue would have received more detailed consideration. It is true that physiological observations of the highest interest are scattered throughout the book, but its general purpose would have been better served had the subject been treated of in more concentrated form. Physiology should have been systematised equally with anatomy. The significance borne by the nucleus in the autonomy of the nerve-cell is worthy of all consideration; but are there not other matters deserving of prior attention. The question of vaso-motor action in relation to the cerebral vessels is not considered; and we have to seek in vain for any detailed observations on the cause of sleep, on cerebral rotation, and on other matters which would have materially assisted the author and reader, both in the clinical and pathological sections.

The clinical section is the result of the experience of four thousand cases occurring in the hospital, which of all others, has done most in promoting the scientific study of insanity; it bears the mark of thorough hospital work, the application of many scientific experimental aids to diagnosis, and of careful recording. The observations on reaction-time, the condition of the blood, and electrical stimulation are especially valuable. The chapters on epileptic insanity, general paralysis, and alcoholic and senile insanity may be regarded as perfect, as least as much so as the present state of knowledge permits. They contain much original matter.

It seems inevitable that authors on insanity must make an effort to analyse disturbances of consciousness from a purely psychological standpoint; and accordingly Lewis, in the chapters on states of depression, stupor, and exaltation, projects a scheme of morbid psychology highly ingenious and seductive, but in our opinion having no bearing on the pathological position, the elucidation of which is the main object of the book. The whole matter is hypothetical, and it would be by no means difficult for a man of like ability belonging to another school of philosophy, to devise a system equally seductive, and equally useless to the physician. We hold with Griesinger that "the entrance to psychiatric medicine is not through the dark portal of metaphysics," and that nervous disease at large, has not been in any way developed "from a specially philosophico-psychological point of view." The psychological method has done much to retard the objective study of the many diseased conditions of which insanity is a symptom. But in this one respect Lewis has followed the beaten track: he "walks the chambers of the (insane) brain,"

and finds no difficulty in tracing out the sequence of psychological events from the incidence of simple morbid depression of feeling, to the most complicated phenomena of delusion, morbid impulse, and mental decadence. The thesis is interesting, here and there vague, especially when attempts are made to collate physiology with mental phenomena, but not harmless: for it leads the author away from consideration of the main issue—the physical causes of implication of the substrata of consciousness. Here he fails us. Early in the chapter on depression he assumes “a state of *cerebral torpor* in the physical substrata of *object-consciousness*, and a state of *cerebral irritation* in the substrata of *subject consciousness* ;”<sup>1</sup> but he fails to offer even a theory as to what produces the torpor or the irritation. The reader is left to infer, in the absence of any further information, that a psychological nexus only exists between the cause and the symptom without the intervention of any direct material changes in the cortical tissues.

What the student desires to learn is in what the primary irritation consists, and how it acts in producing “quantitative and qualitative variations in the nutritive functions of the nervous centres arising from direct disturbance of the blood current in one group of so called varieties of melancholia, and in another induced in the nervous tissues primarily.”<sup>2</sup> (Page 129). The matter, to say the least, is not outside theory. The condition is, so to speak, idiopathic; it begins primarily in the brain, and is due to its over-excitation, whether we regard the assigned causes to be “adequate” or “trivial.” Is it not then worth while to enquire whether the action of the nervous tissues may not produce disturbance of blood supply directly or reflexly. Here we must again remark on the absence of all consideration of vaso-constrictor or vasodilator nervous supply to the vessels of the encephale. Are we to infer that the author agrees with certain physiologists that such systems do not extend to the brain? If so, it should have been stated. This is not the place to express our own views on the subject; but we demur to the opinion that “the very earliest signs preceding genuine pathological depression are really the symptoms of cerebral anæmia and nervous exhaustion.” This we hold to be a traditional assumption, based on psychological

<sup>1</sup> Whilst copying this passage we must enter a protest against the double-leading of words to which importance is attached; it fails to effect the purpose intended by reason of constant repetition, and mars the look of the page. It rather suggests a fulminating psychosis.

<sup>2</sup> The sentence between inverted commas is paraphrased from the text.



considerations, and that physiological, clinical, and pathological data point to the early signs of idiopathic melancholia being symptoms of hyperæmia of the superior convolutions, followed, perhaps, in the later stages by anæmia induced by secondary structural changes in or around the vessels. Lewis, again, alludes to toxæmia as a "qualitative variation of the blood-plasma" inducing depression; but he does not enquire where the change arises, nor alludes to the possibility of the brain supplying its own poison. Where he fails us is in ignoring the extremely difficult question of the patho-genesis of idiopathic disease of the convolutions, produced by over-exercise of function, and symptomatised by mania, melancholia and dementia; he does not go far enough back in his pathology, and takes no account of the physiological work of Gaskell, Foster, and Roy, and little of that of Mosso and Duret, which might have served to add importance to his observations on the vascular unity of the brain in the last section of the book. It is interesting to observe that when we reach such subjects as epilepsy and general paralysis, where the author feels the ground of pathology more firmly under his feet, psychology is discarded, and true clinical work only occupies the page. Were it not that this book is pitched high, the above criticism would be inadmissible.

Conspicuous by its absence is all reference to syphilis as a factor in the production of degeneration of nervous tissue, implying the author's disbelief in its action. In such cases it is as valuable for the reader to know what the author disbelieves as what he believes.

The last section treats almost entirely of morbid anatomy. It is of the highest excellence; no one in Great Britain, save Lewis, could have written it, and we doubt whether on the continent any individual observer could have produced such a series of chapters. Interspersed with the details of morbid histology we find pages characterised by a philosophic pathology, the outcome of deep thought and study, which in subsequent editions will probably be further elaborated. It is hopeless to offer a digest of the contents of this section; the morbid changes found affecting each individual structure are described with a minuteness which to some may appear almost painful, but which is absolutely necessary for the purposes of any student who desires to seek below the surface. The illustrations are delicately executed, and represent with great accuracy the various degenerations. Those who have not examined morbid, or for that matter, healthy brains, by the freezing and aniline-



black staining process, may be inclined to regard many of the pictures as somewhat diagrammatic. This is not so, each illustration delineates with exactitude delicate structures which by this method are readily demonstrable.

There are, however, certain points which demand explanation, and others calling for criticism. In the chapter, for instance, on general pathology and morbid anatomy, in which the appearances presented in all forms of insanity are discussed, certain lesions are ascribed to inflammatory action. The pathological anatomy of chronic alcoholism, epileptic insanity and general paralysis are considered separately, so that we must infer that the general chapter treats of the morbid appearances presented in other forms of insanity, except where the distinction is specially noted. Certain of the changes in bone and membranes are held to be evidence of inflammatory action of old standing; but in the clinical section we find no evidence that the author regards inflammation as a factor in the production of mental symptoms, general paralysis excepted. Where then, and in what forms does it occur? If it is of old standing it must have existed very early in the disease, probably it was the disease itself. If it was not so why did it occur at a later period? "The *pia mater* is abnormally thickened in fully 48 per cent. of those dying insane, partly from fibrinous exudates which have organised, partly from plastic lymph, and often from an œdematous swollen condition of the conjoined soft membranes," &c. This is a fair picture of "bygone inflammatory action," and we must ask the author when and under what circumstances was it existent. Again, in dealing with atrophy of the convolutions, Lewis assumes that all excess of cerebro-spinal fluid is compensatory. No doubt it is so in the very great majority of old standing cases, and in small local atrophies; but in fatal recent cases of acute idiopathic insanity and general paralysis, accumulations of fluid are found which, had they occurred in the subjects of basal meningitis would have been held to be the immediate result of inflammation. Lewis holds strongly that general paralysis is an inflammatory disease, in which opinion we thoroughly concur; that being so, why should it be denied exudation, one of the first and most important of the products of inflammation? which being poured out in quantities larger than the lymphatics and veins can carry off, presses on the convolutions and produces atrophy. We hold that the question is definitely settled by the phenomena presented in an

early case of general paralysis, on which the operation of trépaning was performed at our instance a year ago. A disc was removed from the left parietal bone and the brain did not bulge into the hole appreciably. The same operation was performed at the corresponding spot on the opposite side, and the *dura mater* bulged so much as to occupy the trephine hole. The patient died eight months after the operation from pneumonia: on examination, a large accumulation of fluid was found in the side which had bulged, and there was a corresponding atrophy of the convolutions; on the other side there was little or no fluid, and no atrophy. If the fluid had been compensatory the membrane would not have been forced into the hole. The same has been observed by Dr. Claye Shaw in two cases in which he caused the operation to be performed. Holding the compensatory theory, Lewis is precluded from considering the important influence which is exercised by fluid pressure in the production of symptoms.

The pathological novelty of the book is the theory of the action of the large Deiters, or connective tissue-cells. Lewis holds that the "Lymph-connective" elements of the brain, or "scavenger cells," as he calls them, take a very active part in the processes of disease affecting the nervous centres. In his own words:—"The delicate system of lymph-connective elements permeating in the normal state, the whole of the cerebral mass of white and grey substance, takes a more active share in the pathogenesis of mental decadence than any other; and the more the question is investigated, the greater the importance, we feel convinced, will be attached to these elements in the processes of disease as affecting the nervous centres. Their physiological indications are clear; they are the *scavengers of the brain*; and the evidence obtainable renders it now incontrovertible that they are liable to excessive and rapid development under certain morbid conditions affecting cerebral nutrition and repair . . . . . Whatever leads to increased waste of cerebral neurine, whenever structure disintegration is slowly proceeding, either in nerve cell or fibre; whenever accumulation of *débris* occurs from disease of the vascular tracts, then we invariably note an augmented activity, registered in these scavenger elements of the brain. That their activity is in direct ratio to the functional activity of the essential neurine tissue we think there can be no doubt; nor that with each accession of the nerve-tide they are stimulated to increased activity in the removal of the products of waste and the plasma effused from the vessels.

In healthy states, however, they never assume the hypertrophied form, the deep staining, the coarse fibrillation, the rapid multiplication, and the evidence of obvious inter-cellular digestion, which are readily observed in pathological states." This hypertrophy of the processes being distributed between the nerve elements and surrounding the vascular walls, replaces the delicate neuroglia, and as the cells undergo further alteration they produce a fully formed felt-like material, which destroys the lateral poles of the ganglionic cells, and by ultimate contraction interferes with the permeability of the vessels. Whilst agreeing with the author to a considerable extent, we cannot help thinking that he places undue importance on the action of the scavenger cells on the one hand, and too little on the other; and that we must guard against the theory that there is associated with them in the brain a pathological process different from what occurs in other organs. It is generally accepted that connective tissue cells act as phagocytes in most parts of the body, whether in health or disease. In disease a prominent example is afforded by the connective tissue cells of the lung taking up foreign matter and removing it into the lymph stream, or encapsulating particles. In the early stages of certain cases of catarrhal pneumonia epithelium may be considerably proliferated with but slight apparent increase of connective tissue, which only becomes very marked on the condition becoming more or less chronic. This seems to indicate that the more embryonic (and therefore less easily demonstrable) the connective cells are, the more active is their phagocyte action, and the more they work to remove effete material; only becoming, so to speak, hypertrophied after long subjection to irritation. In other cases the connective tissue becomes rapidly, it may be almost said primarily involved, and the pathological consequences are more serious. We believe the same types of change occur in the brain. In such diseases as general paralysis and chronic alcoholism, the increase of connective tissue resembles that of the latter class of catarrhal pneumonias; in certain other forms of insanity, that of the former, the cell not losing its embryonic character, and therefore acting strongly as a phagocyte, and procurer of restoration to normal conditions. In the diseases mentioned, probably on account of the irritation acting more directly on the spider-cells they proliferate so rapidly as to destroy the protoplasmic poles of the cortical cells by contracting, interfere with the potency of vessels, and by injuring tissues which cannot be reproduced, render recovery

impossible. The strong probability is that in idiopathic and other insanities these "lymph-connective" cells play an important phagocyte rôle, for in that condition there is a large amount of effete matter to be removed, and which, as shown by the large percentage of recoveries, is removed. No one will contend that this is the only agency at work in getting rid of *débris* and devitalised tissue, but there are strong reasons for believing that its influence is considerable. We are inclined to believe that the large easily demonstrable scavenger cells of Lewis are cells which have passed through the more active phagocyte stage, and which, although by no means powerless, have suffered from the over-exercise of function. The reticulum formed by their processes interferes with the healthy relations of tissues, and, as Lewis points out, tends to destroy the association system of ganglionic cell-poles. This reticulum, in fact, constitutes the invariable sclerosis of general paralysis, and the occasional sclerosis found in other forms of insanity. It is a curious fact that where sclerosis is found to have existed, exaltation of feeling has, in a large proportion of cases, been manifested during one period or another of the course of the disease. In general paralysis it is almost invariably present. It is the only definite mental symptom which can be associated with a particular structural change. Can no one offer a theory of the relation of symptoms to lesion?

Lewis asserts that this felting of the processes of the Deiter's cells does not occur in the epileptic insane. Here we differ. In the very last case of this condition which we examined, the proliferation was remarkably well marked, particularly around the larger vessels, and what may be of some importance to notice, the bodies of the large ganglionic cells in the neighbourhood were to all appearance healthy, except that their poles were specially well marked; so much so as to suggest that they were undergoing analogous changes to those of the neuroglia cells. The cell nuclei presented slightly the vacuolation mentioned in the work; but is not this appearance nearly as common in other forms as in epileptic insanity?

Eulogy is thrown away on this book: it speaks for itself in every page; the veriest tyro must see, although he may not appreciate all its merits; and there are very few workers in the same field of enquiry who will not be obliged to admit that Lewis has carried his observations in morbid histology beyond their own range. But much remains untouched even by this book, which

may be said to contain all that is at present known of the morbid appearances presented in the insane. The first new point to be worked out is the locus and period of incidence of structural changes. It is almost a waste of time for the student now-a-days to examine by any very elaborate method the brains of the subjects of old standing insanity; he must apply himself to recent and acute cases which have manifested mental symptoms in any form—coma, delirium, insanity—and to those who have died from head injury. Imitative experiment may also assist. Our belief, founded on an as yet very limited number of observations, is that many changes which have been regarded as secondary are in point of fact primary, and that they are produced with extraordinary rapidity.

JOHN BATTY TUKE.

---

*Injuries and Diseases of Nerves and their Surgical Treatment.* By ANTHONY A. BOWLBY, F.R.C.S. 1 vol., 8vo, pp. 510. London: J. and A. Churchill, 1889.

THIS is the work of a skilled microscopist and accurate clinical observer, and it abounds with facts of the greatest interest and importance. It may be spoken of in terms of highest praise, for after very careful reading we believe that it will take a place beside the classical treatises of Létievant and Weir Mitchell. It marks indeed a distinct advance on the works of these two authors, for not only has Mr. Bowlby been able to avail himself of a literature upon the subject which has much increased since the issue of their books, but microscopical methods have also much improved, and more therefore can be learned of the pathological changes which are the result of nerve injury. The book moreover is not a mere compilation of the work of others; it is full of original matter, based upon the careful observation of an unusually large number of cases in the practice of the Surgeons of St. Bartholomew's Hospital during a period of nine years. And what makes the contents so valuable is the fact that many of the cases have been pursued and observed with unrelenting watchfulness for a long time, so that something might be known as to the ultimate results of treatment in cases

of suture of divided nerves. Nearly half the book is indeed devoted to the subject of nerve suture, and is one long plea in favour of this plan of treatment, supported by the strongest evidence as to its usefulness and good. We commend the lessons therein to all surgeons.

It is our intention in this notice to refer to those parts of the book in which Mr. Bowlby has something new to tell us, or in which his opinions differ from those of previous writers.

As to the regeneration of nerves the importance is pointed out of not relying too much upon the results of experiments on animals, for there seems to be considerable variety in the exact mode both of de- and re-generation in different animals. It is very necessary therefore to trace the process in man. He confirms the observation of Hayem that new nerve elements are to be found in the bulbous end of a nerve divided in amputation, but he goes further than this and gives three cases in which regeneration of the peripheral ends of nerves had occurred after section in continuity independently of union with the upper portion. It is strange that any regeneration should occur without reunion with the nutritive centre, but as to the fact the author has no doubt. This however has to be noted that the regenerative effort is probably short-lived, and that unless union be accomplished degeneration again sets in and may go on to complete atrophy. No stronger argument could be adduced in favour of nerve suture, for it is obvious that connection with the nutritive centre is essential for the continuance and perfection of the process which may have been begun in and by the nerve itself.

Discussing the question of primary union he regards it as quite possible without any antecedent degeneration, for the complete return of sensation and motion is a sufficient proof of it in at least two cases. It is of course very rare, and anything which prevents healing by first intention must oppose the primary union of nerves. The author expresses his firm conviction that the commonly accepted doctrine that *partial* section or wound of a nerve trunk is more dangerous and more likely to lead to complications than complete section is quite erroneous. His experience is moreover opposed to the well-known dictum of Weir Mitchell that "the capacity of nerves to remake their lost parts is almost invincible," for the records of surgery in untreated cases in man tell a very different tale. Nerves whose ends are

separated show no irresistible tendency to unite. If they unite at all they do so in a most imperfect manner and frequently there is no sign of repair.

The chapter on the trophic changes induced by injury is rich in material. He doubts whether glossy skin, as stated by Mitchell, is "never present without burning pain," or causalgia.

Several instances are recorded, with illustrations, of changes in the nails, and in more than one the interesting fact that all the nails of a hand were involved even though the nerve supply of some fingers only had been directly affected. Thus after division of the median nerve, not only may the nails of the index, and middle fingers, and of the thumb show trophic changes, but those of the ring and little fingers may also frequently be affected at the same time and in a similar way. However much may be the median supply to the ring finger it is certain that the little finger has none from this nerve, and the suggestion is offered that the alteration in the nail of the little finger is of a reflex nature.

Arthritic lesions of a mild and chronic kind are in the author's experience amongst the most common results of nerve injury. In from one to six weeks the joints, those of the fingers more especially, will be found stiff and sometimes swollen, painful and tender. Finally there is tolerably firm fibrous ankylosis. This may become permanent in exceptional instances.

He does not agree with Charcot that muscular atrophy occurs most commonly after injuries which are prone to induce a neuritis to which therefore the atrophy is presumably due, and he has no hesitation in saying that in by far the larger number of cases which he has himself seen, and in which there have been trophic lesions more or less marked, the nerve section has been complete and there has been no reason whatever to suspect any neuritis. He believes therefore in the direct trophic influence of the nerves upon the tissues, and that in all probability the same fibres are capable of conveying the different impulses which are generated in the centres.

Supplementary sensations have always been of great and unexplained interest after nerve section. The extent to which anæsthesia is present requires an immense amount of care to estimate, and many recorded observations are probably inaccurate. Nevertheless there are cases in which the retention of a certain amount of sensation has been unquestionably noted after section. In Mr. Bowlby's own experience such supplementary



sensation appears to be most marked immediately after the section and subsequently disappears. One thing is quite certain that a simple return of sensation does not afford sufficient proof of new nerve formation, and that in order to settle this, there must be evidence of improved nutrition; trophic lesions must have passed away; the atrophied muscles must have resumed their normal bulk, their contractility, and reaction to faradism.

Not less remarkable than the trophic changes and the affections both of muscles and joints are the deformities which result, and here the author has something very definite to tell us. He points out that there is no active contraction, as is commonly supposed, in the atrophying muscles and that although they do shrink and grow smaller in every diameter there is not sufficient force in the process to cause actual deformity. The whole secret of it lies in the fact that the muscle shrinks only so long as it is unopposed. If the origin and insertion of a muscle be unduly approximated during the process of atrophy, the muscle remains in that position when the trophic changes have ceased, not because the origin and insertion have been drawn together by such changes, but because the atrophy found the muscle in a certain position and there fixed it. Examples are given in support of this view.

Turning to the practical results of nerve suture we find the author's experience leads him to take a decidedly sanguine view. Although union by first intention is not a thing to be expected, nevertheless the probability is that function will be restored. And here comes the further important conclusion which has been drawn from watching the cases over a long period of time. No definite answer can be given to the question how long it will be before recovery ensues in any given case. It may be weeks months or years. Considerable improvement will probably occur within a year, but the record of cases shows that a much longer time than this may be necessary for the complete restoration of function. No case, he says, should be given up as hopeless until the lapse of at least two or three years from the date of suture, whether primary or secondary, and not even then if any improvement is in progress. Our own experience is in full accord with this opinion. Moreover the nerves of young persons regenerate both more surely and more rapidly than do the nerves of the old, and the prognosis of suture of the musculo-spiral nerve seems better than in the case of the median and other nerves.

We have thus chosen, and have put forward almost in his own



words the views of the author on several points of special interest, and the same might have been done in connexion with other parts of the book, wherein he deals with many topics, to wit: reflex paralysis, the various complications of nerve injuries, the uses of electricity, the respective merits of neurotomy, neurectomy and nerve stretching. Enough however has been said to show the character of the work. The bibliographies which it contains, and excellent plates, very much enhance its value, and we cannot doubt that the book will have a large number of readers, who will derive from it both profit and enjoyment, that it will advance our knowledge of an important branch of pathology, and largely tend to the better and more successful treatment of some very serious injuries.

HERBERT W. PAGE.

## Abstracts of British and Foreign Journals.

**Syphilis.**—Insanity and General Paralysis, By Dr. A. MOREL-LEVALLÉE (*Gazette des Hopitaux*, Oct. 19th, 1889).

The author maintains that there are morbid conditions of brain, due to certain poisons, of which the syphilitic is one, which deserve the name of pseudo-general paralysees, because 1stly, they differ from general paralysis in pathological anatomy; 2ndly, they differ from it also in some points of the evolution of the disease, more especially in the frequency of remissions, and even in their curability, but he admits the difficulty of differentiating the pseudo from the true general paralysis. Unfortunately the name syphilitic-pseudo, general paralysis, has been applied to two series of clinical facts having no pathological connection with one another, dependent upon (*a*) separate syphilomata, (*b*) a diffused periencephalitis attributed rightly or wrongly to syphilis. The author enters into a history of opinion as to the influence of syphilis in producing mental disease and specially general paralysis; he believes that syphilis is an important cause of general paralysis, but whether it should be regarded as a predisposing or an exciting cause, pathological anatomy does not permit us to say.

**Essential Myopathies.** By Dr. RAYMOND (*Gazette des Hopitaux*, October 6th, 1888).

The author considers as an essential myopathy, every amyotrophy in which the muscular fibre seems primarily diseased, and presents an alteration which appears not to be referable to nervous trouble. But it is difficult to exclude the influence of functional disturbance of the nervous system.

Essential amyotrophies may be divided into two groups. 1. Local atrophies confined to a single group of muscles, or even to a single muscle. 2. Progressive primary amyotrophy. Those in the first group are not due to any material lesion of the nervous system, but probably depend upon some functional disturbance of the trophic centres, which, however, is purely hypothetical. In

the second group the intervention of the nervous system is much less probable, and we may believe that the atrophy is due to a primary change in the muscular fibre.

The type facio-scapulo-humeral appears usually in childhood, and first attacks the facial muscles. Besides its peculiarity in picking out the muscles implied by its name, it is distinguished by important characteristics. Fibrillary contractions are absent. Electrical contractility is modified quantitatively, but not qualitatively; there is no reaction of degeneration. The tendon reflexes are preserved. Muscular shortening occurs. The affection progresses slowly; the muscles of the special mechanisms are not involved. The disease is hereditary, and may be a family one. The physiognomy is characteristic. In the single case in which an autopsy was made, absolute integrity of the nervous system was found. The juvenile form of progressive muscular atrophy of Erb has many resemblances to this type, but differs in the absence of facial implication, and in the presence of accompanying hypertrophy of certain muscles, often, however, followed by atrophy. There are close resemblances between this type and pseudo-hypertrophic paralysis, and they may be regarded as varieties of the same disease, which probably includes also the hereditary form of progressive muscular atrophy of Leyden. The fact that these varieties are linked together by transition forms, is a strong argument that they are really one disease.

Beyond heredity no cause is known. No lesion of the central or of the peripheral nervous system has been discovered by the most careful examination, but many observers believe that the disease depends upon a functional disturbance of the trophic centres.

**The Mental Troubles of Old Age.** By Dr. ROUILLARD (*Gazette des Hopitaux*, July 13th, 1889).

In speaking of premature senility, the writer points out that it is sometimes susceptible of amendment, and must be distinguished from true senility, which always goes on to a fatal termination. Still more necessary is it to distinguish the pseudo-senility of young men. In  $\frac{9}{10}$  of the cases premature senility must be attributed to atheroma. Apart from this morbid condition we find it only in melancholics and cachectics.

The first symptom of senile dementia is failure of memory. The memory is destroyed little by little, layer by layer. The failure at first seems slight, and is most marked for recent events. The memory for names, and especially for proper names, is the

first to go. The other faculties undergo an analagous deterioration. The will is almost destroyed. The moral and affective faculties are not less impaired; there is exaggerated emotionalism.

As complications of senile dementia are mentioned—delirious senile dementia, *i.e.*, senile dementia accompanied by delusions and hallucinations—delusions of persecution. These differ from the same delusions in delusional insanity in not being coordinated, and in being multiple, changeable and fugitive. Hallucinations present the same characters as the delusions. They are not always disagreeable. Visual hallucinations are the most common. Illusions, misrepresentations of sense impressions are still more common. Melancholic and hypochondriacal delusions; of these the physical condition is the most powerful factor. Patients may appear melancholic who are really not so. Religious delusions; their presence makes the prognosis more unfavourable; erotic ideas; ideas of grandeur.

In old men indecent acts are usually prodromata of dementia they often occur before there is obvious mental failure.

*Morbid Anatomy.*—Two facts dominate senile dementia arterial atheroma and brain atrophy. Atrophy affects the cerebrum more than the other parts of the brain, and especially the left hemisphere and the temporo-parietal region. The writer does not share in the views of Luys, who regards loss of weight as much more important than atheroma.

**Schneider on Nasal Reflex Epilepsy** (*Berl. Klin. Woch.*, No. 43, 1889).

The author relates six cases which were cured by treatment of the nasal abnormality.

I.—Male, æt. fourteen years, without hereditary taint; epileptic since the age of five years. Fits occurred nightly while falling asleep or shortly after commencement of sleep. Often an asthmatic attack immediately preceded a fit. Both nares were almost occluded by polypoid swellings; a large mucous polypus hung from an unciform exostosis of the septum. Touching the inferior turbinate swellings of either side educed sibilant bronchial râles and cough, with great anxiety of facial expression, followed in the course of half an hour by a severe fit. The patient has had no epileptic or asthmatic seizure since the morbid growths were removed in November, 1885.

II.—Female, æt. twenty-four, no heredity; epileptic from the time of puberty; subject to vertigo and migraine. For some

years received benefit from bromide. The origin of the epilepsy was then (July 1885) accidentally disclosed by the occurrence of a severe fit which followed an attack of sneezing brought on by looking towards the sun. The nasal mucous membrane was found to be greatly tumefied; reflex cough was excited by contact of various points. On the right inferior turbinate was a spot, irritation of which caused mydriasis of the same side and exclamation of feeling ill. The mucous membrane was brought into a healthy condition by cauterisations, since which the patient has been free from epilepsy, vertigo, and headache.

III.—Absence of fits since the tumefied nasal lining was reduced by the cautery two years ago.

IV.—A strongly built man, free from heredity, æt. thirty-five, in 1885 suffered from frequent dry cough, dyspnoea, paroxysms of asthma with vertigo and “flying heats,” also from occasional convulsive attacks with loss of consciousness. Rhinoscopy shewed a small exostosis surmounted by a soft tumour, the size of a large pea, on the right lowest turbinate. When this tumour was touched patient began to cough, complained of irritation beneath the sternum, and pain in the back; sibilant dyspnoea, extreme anxiety of expression, and dilatation of right pupil ensued; patient started up with a scream, stood rigid for a moment, then fell in a fit. Immediately after removal of the soft tumour (an angioma in which were many nerve filaments) the patient expressed complete relief from all his symptoms. Was immune for a year; occasional vertigo then developed and recurrence of the soft growth was discovered. This and the exotosis were extirpated. So far, perfect recovery has resulted.

V.—Male, æt. forty, no heredity; has been under treatment for two-and-a-half years on account of various nervous troubles; had suffered from epilepsy for some months; complained of severe asthmatic paroxysms. During even slight mental excitement convulsive tic, blepharospasm, and salaam convulsion appeared. Several polypi were found attached to each inferior turbinate bone; since they were destroyed (two and three quarter years ago), all the convulsive and asthmatic seizures have been absent.

VI.—Female, æt. seventy-two, began to suffer from mimic convulsion, nystagmus, and severe blepharospasm after the occurrence of a nasal catarrh. Attacks of unilateral clonic wry-neck became established and gradually developed into *le haut mal*. On the right lowest turbinate there was a large soft tumour, cauterization of which (two years ago) was followed by an immediate and entire cessation of all the nervous symptoms.

In one case of long-standing epilepsy with nasal polypi this treatment has failed.

**Schæfer on Railway-spine** (*Ibid*, No. 43).

A case is cited to show that the neuropsychosis designated as above may result from mental shock alone. The patient, a strong temperate man, while driving his engine round a curve suddenly observed the three red lights of a train that was standing on the same line of rails fifty metres in front of him. He stopped his train in time to prevent a collision, but during the moments of danger he suffered violent mental agitation, producing uncontrollable tremor of his legs. In the course of a few days he utterly broke down and rapidly emaciated; gait was trailing and laborious; speech difficult and stammering; hands trembling; tongue showed coarse and fibrillar tremor; pulse was quickened to 104; reflexes exaggerated in a moderate degree; Romberg's symptom present, also much general muscular enfeeblement. Disposition excitable and melancholic; eyes red from weeping; patient sought solitude, displayed great fear, had frequent vertiginous attacks and constant severe headache. Memory greatly impaired. Sleep much disturbed by terrifying dreams. Very marked diminution of cutaneous sensibility; a needle prick sufficient to draw blood was scarcely felt as a mere contact sensation. Subjectively the legs felt cold, numbed and paralysed, and there were frequent flashes of light before the eyes. Pupils equal and re-acted well. Anorexia; taste disordered. Involuntary micturition.

E. BIRT.

# BRAIN.

PART III., 1890.

## Original Articles.

### SYRINGOMYELIA.

BY DR. PAUL BLOCQ.

*Chef des travaux Anatomo-Pathologiques à la Salpêtrière.*

*Translated from the original MS. by H. W. Marett Tims, M.B. (Edin.).*

IT is not uninteresting to notice how, as neuro-pathology advances, careful clinical observation, aided undoubtedly by the support which pathological anatomy gives to it, and often not less aided by the data of experimental physiology, clinical observation, I say, succeeds in separating, from a group apparently homogeneous, a certain number of special and clearly differentiated nosographical types, until, after a series of segregations, the original group is reduced to almost nothing.

This evolution, of which the study of the paraplegia of former days affords a noticeable example, is not less evident in the history of progressive muscular atrophy.

The morbid type, described by Duchenne-Aran, originally included all the known forms of progressive muscular atrophy. Soon afterwards Duchenne (of Boulogne) himself distinguished pseudo-hypertrophic paralysis, which he knew to be of muscular origin, and which he put in a separate division. Later on Charcot, in his turn, differentiated lateral amyotrophic sclerosis, the peculiarities of which he pointed out. Landouzy and Déjerine soon showed the myopathic nature of hereditary muscular atrophy, and they, too, singled it out. Then Erb, Leyden, Von Eichorst, have each distinguished different varieties, and separated them from the

original group of progressive myopathies. And now, quite recently, syringomyelia has been differentiated, which, even during the last few years, has been very often confounded with progressive muscular atrophy, certainly in its clinical aspects.<sup>1</sup>

# I.

*Historical.*—If the preceding considerations are to a certain extent a summary of the clinical history of the malady under consideration, this is far from being the case as regards the anatomical history, for on this point the chief lesion has been recognised for some time past. Starting from these two points, one might divide the history into two phases; (i.) an anatomical period, during which the disease was only studied *post-mortem*; and (ii.) a clinical period, in which the symptoms depending on the lesion were made out.

(i.) The word syringomyelia (συριγγώδης, hollowed out in the shape of a pipe; *μυελός*, marrow) was used for the first time by Ollivier (d'Angers).<sup>2</sup> This author did not admit the existence of the central canal in the spinal cord, and he considered that the different facts brought forward in support of its existence, by Brünner,<sup>3</sup> Santorini<sup>4</sup> and Rachetti<sup>5</sup> were to be attributed to an arrest of development. Ollivier, of Angers, can only then be credited with the invention of the new term, and not with any real idea of the disease which the term indicates, and of which he makes no mention.

When subsequent anatomical researches had incontestably

<sup>1</sup> The following passage, to be found in the *Symptomatologie of Muscular Atrophy in the Adult* (De l'électrisation localisée, p. 493), seems to show that Duchenne has made this confusion:—"The anæsthesia," he writes, "is sometimes so marked, that patients are neither sensible to the strongest faradic excitations nor to the action of fire. I have seen some of them allow the anæsthetic areas to become extensively burnt, owing to their not having felt the effect of the fire, and not having seen that the parts were in contact with it." Moreover, we have lately seen at the Clinique an old-standing case of atrophy, as recognised by Duchenne, diagnosed by M. Charcot as a case of syringomyelia. There are thus classified in the wards of the Salpêtrière as disseminated sclerosis, various cases which on further examination, and in one case a *post-mortem* examination, were recognised as syringomyelia.

<sup>2</sup> Ollivier. *Traité de la moelle épinière et de ses maladies*, 1827, t. i., p. 178.

<sup>3</sup> Brünner. *Vide* Morgagni. *De sed et caus. morb.*, ep. xii., sect. ii.

<sup>4</sup> Santorini. *Advers. anat.* vi., Obs. xiv., pp. 17 et 18.

<sup>5</sup> Rachetti. *Della struttura della medole spinale*, Milano, 1816.



proved the normal existence of the central canal in the cord, syringomyelia was soon forgotten.

It was not long, however, before observations of the abnormal dilation of this canal were made, and the majority of them were at the time looked upon as arrests of development, and the name hydromyelia was commonly given to them. Nevertheless, certain observations treated of lesions which it is difficult to attribute to a congenital anomaly; such, amongst others, are the facts recorded by Gull,<sup>1</sup> Mayer,<sup>2</sup> and Schüppel.<sup>3</sup> M. Hallopeau<sup>4</sup> believed that cases such as these ought no longer to be classed under the heading hydromyelia, but that they should be described under the name of "Diffuse Peri-ependymal Sclerosis." This form of myelitis was, in fact, characterised by the presence of groups of separate cavities in the spinal cord, due to an inflammatory sclerosis of the neuroglia, of which these cavities constitute one method of development.

About the same time Charcot and Joffroy<sup>5</sup> published a case of a similar nature, but they supposed the formation of these cavities to be due to a granular disintegration (the granular disintegration of Clarke). After this comes a series of anatomical works, the first of which is that of Grimm.<sup>6</sup> These tend to show the old syringomyelia, afterwards designated hydromyelia, then peri-ependymal myelitis, to be in reality a neoplastic lesion. The opinion of Grimm, according to which it is a gliomatous tumour, developed at the expense of the elements of the central canal, is adopted in turn by Simon,<sup>7</sup> Westphal,<sup>8</sup> and Leyden.<sup>9</sup>

(ii.) This affection did not really take a definite place in

<sup>1</sup> Gull. Cases of Paraplegia. *Med. Chir. Transact.*, 1856.

<sup>2</sup> Mayer. *Die Electricität in ihrer Anwendung auf Practic. Med.* Berlin, 1861.

<sup>3</sup> Schüppel. *Ueber Hydromyelus.* *Arch. der Heilkunde*, Leipzig, 1865.

<sup>4</sup> Hallopeau. *Contribution à l'étude de la myélite diffuse péri-ependymaire.* *Gazette Médicale de Paris*, 1870.

<sup>5</sup> Charcot et Joffroy. *Deux cas d'atrophie musculaire progressive avec lésions de la substance grise et des faisceaux antéro-latéraux de la moelle épinière.* *Archives de Physiologie*, 1869.

<sup>6</sup> Grimm. *Ein Fall von Progressiver Muskellatrophie*, *Virchow's Archiv*, 1869, Bd. 48, p. 445.

<sup>7</sup> Simon. *Arch. f. Psychiatrie*, t. v., 1874.

<sup>8</sup> Westphal. *Idem.*

<sup>9</sup> Leyden. *Virchow's Arch.*, 1876.

the description of diseases until the works of Schültze,<sup>1</sup> professor at Dorpatt, and Kahler,<sup>2</sup> of Prague, who have established—as M. Charcot<sup>3</sup> proclaimed in one of his lectures—that a certain number of particular symptoms might be traced to that organic lesion, and might be permitted even to determine the principal peculiarities concerning the seat and extent of syringomyelia.

It is, however, necessary to notice that M. Charcot,<sup>4</sup> as early as 1874 had pointed to syringomyelia as one of the possible causes of muscular atrophy of spinal origin. Other very important clinical and anatomo-clinical works have appeared of late years; the memoirs of Fürstner and Zacher,<sup>5</sup> of Bernhardt,<sup>6</sup> of Oppenheim,<sup>7</sup> and the thesis by Mdle. A. Bäümle.<sup>8</sup> MM. Joffroy and Achard<sup>9</sup> have published a paper on the anatomy and pathogenesis of this malady, which will be referred to subsequently. Finally, a communication by W. Roth, of Moscow, in the *Archives de Neurologie*,<sup>10</sup> constitutes a most interesting and complete document on this subject.

MM. Debove and Déjerine<sup>11</sup> were the first to bring the clinical aspects of syringomyelia into prominence by showing patients suffering from this disease at the Société Médicale des Hôpitaux.

Within the last two years works on this subject have appeared in rapid succession. The whole question has been renewed, so to speak, by publications appearing simultaneously in different countries. We will single out for special mention the masterly lecture by Charcot,<sup>12</sup> the paper by Déjerine,<sup>13</sup> and the thesis of Brühl<sup>14</sup>; the last mentioned, written under

<sup>1</sup> Schültze. *Idem*, 1882.

<sup>2</sup> Kahler. *Prag. Med. Wochens*, 1888.

<sup>3</sup> Charcot. *De la syringomyélie (leçon recueillie par M. le Docteur Paul Blocq)*, *Bulletin Médical* Juin, 1889, et *leçons du mardi à la Salpêtrière*.

<sup>4</sup> Charcot. *Leçons sur les maladies du système nerveux*, t. ii., p. 216.

<sup>5</sup> Fürstner et Zacher. *Arch. f. Psychiatrie*, 1883.

<sup>6</sup> Bernhardt. *Berlin Klin. Wochens.*, 1884, et *Centralbl., f. Nerven-heilk.*, 1889.

<sup>7</sup> Oppenheim. *Charité Annal*, xi., 1886.

<sup>8</sup> Mdle. Bäümle. *Thèse de Zurich*, 1887.

<sup>9</sup> Joffroy et Achard. *Archives de Physiologie*, 1887.

<sup>10</sup> W. Roth. *Archives de Neurologie*, 1887, 1888 et 1889.

<sup>11</sup> Déjerine. *Société Médicale des Hôpitaux*, Février, 1889.

<sup>12</sup> Charcot. *Leçons du Mardi*, 1888-1889, 21st *Leçon*.

<sup>13</sup> Déjerine. *Bulletins de la Société de Biologie*, 1890, 9 *serie.*, t. ii.

<sup>14</sup> Brühl. *Contribution à l'étude de la Syringomyélie. Thèse de Paris*, Ferrier, 1890. (*Delahaye et Lecrosnier* edit.)

the guidance of M. Debove, contains the most complete bibliography on the subject.

## II.

*Division. Definition.*—Syringomyelia comprises, etymologically speaking at least, the study of all the affections in which cavities in the spinal cord are to be observed.

We have, however, seen that cases in which the cavities are of congenital origin are to be excepted, and are included in the term hydromyelia.

It remains then to consider syringomyelia, (1) as the result of myelitis, and (2) as the result of glioma. Some authors affirm that cases coming within the first category ought, without doubt, to be placed in the second. M. Charcot, quoting observers who have described cases of myelitis accompanied with the formation of cavities, thinks that it is not possible to admit this confusion, and while remarking that that form of syringomyelia due to glioma had alone been, up to the present time, diagnosed during life, he described it exclusively in his clinical lecture.

Believing that there might easily arise in this discussion, a divergence simply concerning the interpretation of a lesion which is at the same time neoplastic and inflammatory and fearing to prejudge the matter, we adopt the following definition:—*Syringomyelia is a chronic affection of the spinal cord, characterised anatomically by cavities formed pathologically in this organ, and clinically by certain alterations in the sensibility, associated with trophic disorders.*

## III.

*Pathological Anatomy.* — Syringomyelia comprises a special principal lesion, with common secondary lesions; the former being situated in the spinal cord, the latter in different tissues. These consist of trophic alterations in muscle, bone, cellular tissue, skin, &c., but in these situations there are no special peculiarities, consequently we shall turn our attention principally to the alterations in the spinal cord; but we shall have to consider the recent researches which

have been made in the peripheral nerves by MM. Holschewnikoff and Déjerine.

(i.) *Macroscopic*.—After opening the spinal canal and making an incision through the *dura mater*, the cord at once presents in most cases an abnormal appearance, which can be better studied when it is removed from the bony canal. As a rule, the meninges present a healthy appearance, for it is relatively rare to notice a slight amount of posterior meningitis, consisting of patches of thickening of the *pia mater*. If the lesions in the cord are very well marked, it presents the appearance, in the parts most affected, of a large blood vessel, empty and collapsed. This comparison was fully borne out in a case we have just had the opportunity of examining, in which this condition of the cord was present from the cervical enlargement to the level of the lumbar region where the organ was again normal.

In ordinary cases the cord is only irregularly increased in size, and deformed in places.

On palpation, an alteration in the consistence of the cord can be easily detected; it is soft, and there is well-marked fluctuation. If, on the other hand, the cavity be very small, one feels a hard firm cord giving the sensation of a rigid stalk, imbedded in the cord; this consistence belongs precisely to glioma, which in these cases constitutes a veritable tumour.

If a transverse section of the cord be made, one or more characteristic central cavities are visible to the naked eye. As a rule, only one cavity exists, but at times there may be two or three, entirely separate from the central canal. These lacunæ are always situated in the grey matter—an important point to notice—their position here in order of frequency being, according to Mdlle. Baümle, the two posterior horns, the two anterior horns, and any one of the four horns indifferently. The lesion is, as a rule, bilateral.

The size of the cavity varies; sometimes it is reduced to a simple slit, at others it occupies the whole extent of the cord of which only sufficient remains to form the thin wall

of the cavity. There are all gradations between these extremes.

The extent of the affected area may likewise vary in the longitudinal direction. It may occupy the whole length of the cord (Schültze) and may encroach upon the medulla. The ascending root of the trigeminal, the olivary body, the nucleus of the hypoglossal may be invaded. It may also extend as low as the *filum terminale*.

The cervical enlargement is most frequently affected, and then it extends upwards for several centimetres and in a downward direction, throughout the entire dorsal region, leaving the lumbar portion of the cord unaffected.

The direction of this pathological canal is not absolutely straight, it is more or less bent upon itself and may be prolonged into sinuses, resulting in the formation of an irregular-shaped cavity. More often it is elongated in a transverse direction, at other times the central part is constricted and the cavity enlarged at each side, like an hour-glass. Or it may be heart-shaped, pear-shaped, or rectangular with the angles rounded off, or indeed, the shape may be indescribable.

The contents of the cavity are usually liquid, like the cerebro-spinal fluid, but it varies sometimes in colour and consistence, occasionally being almost gelatinous. The lining of the cavity is marked by a kind of smooth yellowish, membranous cuticle, easily seen by the naked eye, to the structure of which we shall again refer. Sometimes small papillæ are present on the surface. The adjacent parts of the cord are necessarily affected in varying degrees, according to the position and extent of the lesion. It is thus that compression or destruction of the grey matter and of the columns of white matter is to be seen. As to the central canal it is usually unaffected, it may be displaced in front of the cavity, its form more or less altered, or it may be bisected. Occasionally it communicates to a certain extent with the pathological cavity.

(ii.) *Histology*.—According to the majority of authors the microscopic changes consist in a neoplastic hyperplasia of the neuroglia of the grey or gliomatous matter; accord-

ing to other authorities, in an inflammatory hyperplasia or myelitis.

The chief seat of these changes is the grey matter, where it is chiefly found around the central canal, and in the gelatinous substance of Rolando, which are of a similar structure. The tissue of new formation, or neoplasia, which constitutes it—the nature of which we shall not here discuss—is formed of a yellowish-brown tissue and of a consistence so firm that it can by these characteristics be easily distinguished from the substance of the cord. The mass chiefly results from an agglomeration of large cells with processes—spider-like cells—which are more or less heaped one on the other, and send out branches which anastomose with each other and thus enclose minute spaces.

The cells themselves consist of a small mass of granular protoplasm with one or two nuclei situated either in the centre or at the periphery and stain well with the ordinary re-agents.

The trabecular processes are slender, rather long, and sometimes show slight enlargements at the point where they unite with each other. The network formed by these anastomoses is rather dense, but nevertheless it encloses, as we have said, small spaces, which contain indistinct granular elements, with free nuclei and some very slender fibrils. Finally, there exists in this hyperplastic mass, a large number of pigment granules and of small, ill-defined, rounded, yellow, refractile bodies, either isolated or in groups, to which the colour of the tissue is due.

The limiting layer which, as we have seen, is visible to the naked eye, is formed of a yellowish membrane, continuous on its outer aspect with the tissue we have been describing. The inner surface, generally smooth, sometimes presents prolongations given off in the form of buds, which more or less divide the cavity. This membrane is essentially formed by a dense fibrillary felting not unlike the conjunctival network. It differs from it, however, in that it is apparently formed by the trabecular prolongations of the cells peculiar to the glioma of which it appears to be a thickening. It is therefore, notwithstanding appearances, no question of

sclerotic tissue. According to some writers this cavity is only lined by cylindrical epithelium, which has been described in certain conditions, when the cavity communicates with the central canal of the cord. It is to be added that this reticulated tissue contains a network of numerous capillaries.

One can conceive that the parts of the spinal cord adjacent to this gliomatous hyperplasia are more or less affected by it. As a matter of fact, the different elements of the cord are variously affected. They are either compressed as the lesion extends, or separated out by an infiltration of the neuroglia, and are consequently displaced or undergo alterations. In the latter case hæmorrhage, inflammation and degeneration may occur. The first two of these methods characterise the ordinary reactions of the grey matter; the last is more peculiar to the white matter. Sclerosis of the white columns results, with subsequent ascending or descending degeneration of the columns so affected. These secondary lesions present, as we shall see, the greatest interest from a clinical point of view.

*Peripheral Nerves.*—Recently attention has been turned to the examination of the condition of the peripheral nerves in syringomyelia.

Holschewnikoff<sup>1</sup> was the first to show in a case of this affection that the posterior roots of the cervical nerves lose their myeline, and would not stain by Weigert's method. Moreover, the nerves of the Brachial plexus and of the periphery in the limbs shewed hyaline patches. M. Déjerine<sup>2</sup> has systematically worked at the alterations in the cutaneous nerves in an autopsy, and has communicated his results to the Société de Biologie. The intra-muscular nerves of the muscles that had not undergone atrophic changes were found to be normal; but, on the other hand, the intra-muscular nerves of the muscles that were atrophied were very much altered, and were only represented by the empty sheaths. The cutaneous nerves had undergone changes in those parts of the skin where the phenomena of dissociated sensibility existed.

<sup>1</sup> Holschewnikoff. *Archiv. de Virchow.* Fasc. i., p. 10 (1890).

<sup>2</sup> Déjerine. *Loc. cit.*



In this situation half of the large tubes had disappeared, and were represented only by empty sheaths. Smaller tubes which stained badly with osmic acid were very numerous. Tubes undergoing changes were rare—a fact which clearly shows the slowness of the neuritic change in this case.

#### IV.

*Pathogenesis.*—What is the nature of this process? and how does it lead to the formation of the cavities? The various pathogenic explanations which writers on this subject have devised, correspond with the idea that each is of the nature of an alteration, from which it follows that these two questions are identical.

M. Brühl<sup>1</sup> has very accurately pointed out the various theories without venturing to adopt any one of them. Let us pass them in review, following in the lines of his excellent work.

According to some, Langhans,<sup>2</sup> Stadelmann,<sup>3</sup> Steudener,<sup>4</sup> the origin of the cavity is vascular; it is a question either of œdema of the tissue around the central canal, or of hæmorrhage with subsequent reabsorption, or of Colloid degeneration of the vessels.

Others, Eickholt,<sup>5</sup> Hallopeau,<sup>6</sup> Silcock,<sup>7</sup> Joffroy and Achard,<sup>8</sup> see in it an inflammation, a myelitis. The cavity is then caused by the contraction of the sclerosed perpendymal tissue, the destructive tendency of the hyperplasia, or by softening and breaking down consequent upon thrombosis of the vessels in the inflamed area.

Lastly, according to the majority, Simon,<sup>9</sup> Westphal,<sup>10</sup> Roth,<sup>11</sup> Mdle. Bäumlér,<sup>12</sup> the degeneration of the neoplasm, the glioma, is the sole cause.

But what is this tissue of new formation? Is it the production of an inflammatory hyperplasia, as the partisans

<sup>1</sup> Brühl. *Loc. cit.*                      <sup>2</sup> Langhans. *Arch. de Virchow*, 1881.

<sup>3</sup> Stadelmann. *Arch. f. Klin. Med.*, 1883.

<sup>4</sup> Steudener. *Hirsch's Jahresbericht*, 1867.

<sup>5</sup> Eickholt. *Arch. f. Psychiatrie*, 1880.

<sup>6</sup> Hallopeau. *Loc. cit.*

<sup>7</sup> Silcock. *Brit. Med. Journ.*, 1888.

<sup>8</sup> Joffroy et Achard. *Loc. cit.*

<sup>9</sup> Simon. *Loc. cit.*

<sup>10</sup> Westphal. *Loc. cit.*

<sup>11</sup> Roth. *Loc. cit.*

<sup>12</sup> Mdle. Bäumlér. *Loc. cit.*



of the theory of myelitis would have it, or is it a neoplastic hyperplasia, as those who support the glioma theory affirm?

On this point we are disposed to endorse the following hypothesis of Schültze,<sup>1</sup> which, as we shall show, would reconcile these opinions, apparently so at variance. The new tissue would be in reality both neoplastic and inflammatory. If, on the one hand, simple inflammation of an ordinary tissue, for example the conjunctival, assumes the characteristics of proliferation of the nuclei, and is terminated by sclerosis (as happens in the cord, particularly in the white matter, the sclerotic inflammation of which has most frequently its point of departure in the connective tissue of the vessels); on the other hand, inflammation confined to a special tissue, as the neuroglia, has then its particular characteristics of gliomatous proliferation, and leads to disintegration and the formation of cavities. Schültze would naturally call this special kind of inflammation *gliose* or *gliomatose*. A neoplasiform inflammation would exist, then, in the spinal cord in syringomyelia, not on account of any particular characters of its own, but by virtue of the tissue in which it has its origin. It is on this account, in our opinion, that writers have looked upon the lesions in the light of a myelitis or of a neoplasm, according as they considered its characters as being chiefly inflammatory or chiefly neoplastic.

This eclectic and original idea finds a firm foundation in the recent works on the embryology of the brain and spinal cord, which show that the conjunctival membrane arises from the mesoderm, the neuroglia from the ectoderm, and, consequently, the pathological changes should be different.

Under these circumstances, it becomes a case, then, of a slow proliferation of the neuroglia, resulting in the formation of the so-called gliomatous tissue. This tissue, as it gives rise to a number of tissues of pathological origin, would soon present phenomena of disintegration, either from disorders of the circulation, or from the simple fact of the

<sup>1</sup> Schültze. *Loc. cit.*

natural tendencies of the tissue, and a cavity would thus make its appearance. To resume:—these data being admitted, if the name *gliomatose* is applicable to the lesion, that of *cavitary myelitis* (*myélite cavitaire*), lately proposed by MM. Joffroy and Achard, if one were to prefix the qualification *neuroglic* (*névroglique*), would not be out of place.

## V.

*Etiology.*—Very little is known about the causes of syringomyelia. It must certainly be less exceptional than one might suppose, and its rarity arises, doubtless, from its having been little diagnosed till quite recently. We have had the opportunity of seeing six cases in one year in the Salpêtrière.

The age at which the disease first begins to show itself varies between fifteen and thirty-five years. Statistics show men are attacked more frequently than women, in the proportion of three to one. We have collected but few references to any hereditary nervous tendencies. One can include amongst the number of determining causes the majority of debilitating affections which are generally present at the development of other nervous diseases—injury, overwork, chills, infectious diseases, drink. It is also sometimes the case that not any of these influences are discoverable in the previous history of the patients.

Consequently, one is disposed to think that the cause lies in some embryological defect. It would be a malady due to a defect of evolution, upon which the chance causes we have mentioned might have had but an accidental effect by interfering with the nutrition of the nerve elements, and consequently diminishing their power of resistance to the hyperplastic tendency inherent in the neuroglia.

## VI.

*Symptomatology.*—There are great varieties in the symptoms of syringomyelia, but nevertheless, we will commence by describing one of mean type, fitted to give a general idea

of the condition, reserving till later the more particular symptoms, and the construction which pathology allows to be put upon them; and lastly, some of the plans by which we think observations may be grouped and classified.

(i.) The disease commences insidiously, usually by increasing weakness in the upper limbs; pain, properly speaking, is rare, but a sensation of numbness may be present in these limbs. Soon muscular atrophy is added to the functional weakness. Sensory disorders, which may have existed from the commencement, become marked. Then spinal curvature, in the form of scoliosis, becomes a symptom; and finally, various motor troubles attack the lower limbs.

Examination of the patient at this stage shows the following points:—First of all, one notices his peculiar attitude, due to the curvature of the vertebral column, and one is struck by the claw-like deformity of the hands, similar to the condition so often seen in progressive muscular atrophy of the type described by Aran-Duchenne. The hands are wasted, cyanosed, and show various trophic changes in the skin and nails; the fingers may be the seat of spontaneous ulcers and painless whitlows. Finally, the loss of muscular power is very noticeable, and the tendon-reflexes are either absent or diminished. If one examines the state of the sensibility, one observes this peculiarity, almost pathognomonic—that, while sensibility to touch is perfectly retained, sensibility to pricking, as well as that to heat and cold, are completely abolished, not only in the upper extremities, but over more or less extensive areas, and sometimes over the whole body.

The various systems, pulmonary, cardiac, gastro-intestinal, are functionally regular. The urinary excretion alone is more or less affected.

Without great modifications, if one excepts the super-vention of various trophic changes, arthropathies, excessive secretion of sweat, &c., and after oscillating between better and worse, the disease slowly progresses and the patient succumbs, often at an advanced age. This event is very frequently due to an intercurrent or secondary cause.

(ii.) Let us return now to the more salient points, which at present we have merely enumerated.

*Sensibility.*—The most important clinical verification of the diagnosis which can be made at the bedside is the pathological dissociation of the different kinds of sensibility.

Three forms of sensibility are affected, viz.:—to pain, heat and cold. Three forms are unaffected—sensibility to touch, the muscular sense, and the special senses. This law thus formulated is not by any means absolute; it would not hold good, for example, in a case in which the sensibility to touch is affected. But this is the exception.

*Thermo-anæsthesia* is very frequently overlooked by the patient, but the physician may be on the look-out for it, where there exists, as is so often the case, many traces of former burns upon the limbs. It is necessary to distinguish sensibility to heat from that to cold, for the anæsthetic zones for each are far from regularly corresponding in all cases. It has been noted that this alteration of the sensibility is one of the first symptoms in point of time. The degree to which it is affected, which can only be correctly estimated by the aid of a special thermometer, varies greatly, not only in different patients but often in different spots in the same patient. Some commence to feel a sensation of heat at 60° cent.: or the entire perception of temperature may be abolished. The variation of sensibility to cold is ascertained by means of water, ice, or freezing compounds. In one case reported by Schültze and in another by Debove, there existed at some points a veritable hyperæsthesia to temperature.

The condition of thermo-anæsthesia is distributed very irregularly over the surface of the body; occasionally every part may be affected, the mucous orifices not even escaping, the eyelids, nose, mouth, urethra and rectum. More frequently it occurs in considerable zones; there may be large patches on the trunk, in the middle of which, in some cases, there may be a sensitive area. The limbs may be affected throughout, or only in segments similar to the condition seen in the cases of hysterical anæsthesia described by Charcot. The head also may be equally subject to this condition of thermo-anæsthesia. It increases in extent and in intensity, usually according to the duration of the disease.

In certain places this thermal anæsthesia may be replaced by a true hyperæsthesia. Brühl has observed this phenomenon in a very marked degree and it has been verified by other writers. It is also to be noticed that this condition of thermo-anæsthesia is not constant as to its distribution, it can in fact vary, in a very limited degree, it is true, from one day to another. Lastly, there may be a true perversion of the thermal sense, cold objects producing a sensation of warmth and warm objects being felt as cold.

*Analgesia* is seen quite as frequently and varies in the same manner as to its intensity and distribution. Thus, it may be absolute, or more or less impaired; it may be general and be present in both skin and mucous surfaces, or it may occupy exclusively certain zones. Not only are pinching and pricking not felt, but various lesions of an inflammatory nature, as whitlows and ulcerations, are painless.

Instead of analgesia there may be hyperalgesia. This, according to Charcot, may be explained by supposing that the period of destruction has been preceded by a period of irritation.

This condition of hyperalgesia has received special attention from Renz and Wichmann.<sup>1</sup>

*Sensibility to touch* persists, as we have said, and this is one of the reasons without doubt why the disease has so long escaped clinical detection. All the sensations of temperature, pricking and even of the slightest contact are perceived as tactile impressions and are readily localised. This is also the case with the muscular sense. The patient appreciates the sense of weight and can readily describe the different positions into which the limbs may be placed, and the slightest movements affecting them.

*The special senses*, sight, hearing, taste, smell, remain equally unaffected in spite of the analgesia of the mucous surfaces of the special organs. These are negative characters, the importance of which M. Charcot has pointed out.

Certain painful phenomena also arise from these sensory alterations. Sometimes it is only a question of heat and cold, at others the patients complain of actual pain, which is

<sup>1</sup> Wichmann, Geschwulst und Höhlenbildung ins Rückenmark, 1887.

often but slight and may consist of cephalalgia, pain in the spine and in the joints, and it may also happen sometimes that they may take on the definite character of lightning pains, very like tabetic crises.

*Motility.*—Disorders of motion are only secondary, except in so far as it appertains to the permanent paralysis of the upper extremities.

They consist of paraplegia, rarely complete, of the spasmodic kind, or of inco-ordination of the lower limbs.

Following one or other of these, the patella reflexes may be either exaggerated or abolished. In addition to these motor troubles, it remains to mention the peculiar shaking or trembling sometimes observed.

*Scoliosis.*—Scoliosis, which is almost a constant feature of syringomyelia, has its place here, between the consideration of the motor affections and the trophic changes which follow; for, according to various authorities, it would depend on one or other of these. It makes its appearance at a relatively early stage and attacks the dorso-lumbar region and has its convexity to the left, thus differing from the ordinary scoliosis, which occupies the cervico-dorsal region and has its convexity to the right. It necessarily brings about secondary deformities, which are essentially mechanical.

*Trophic Disorders.*—The most constant of these are in the muscles and are of the nature of atrophy. It takes place in the hands, where it may attack the muscles supplied by the radial nerve, or it may attack those supplied by the median and ulnar, where it brings about the peculiar claw-like condition of the hands. The atrophy afterwards extends slowly and symmetrically to the fore-arm, arm and trunk, resulting in deformities like those caused by other progressive myopathies.<sup>1</sup> The muscles generally show fibrillary twitchings peculiar to muscular atrophy of spinal origin; and in some cases upon electrical examination, the muscles give the re-action of degeneration. The electrical excitability is

<sup>1</sup> Nevertheless, the distribution of the muscular atrophy is here, at least in the early stages, essentially different from that seen in primitive myopathies. In the latter case, the embryological muscular territories (Babinski-Onanoff) are attacked, whilst in syringomyelia, it is the nerve territories.

usually only diminished. According to certain observations, the atrophy commences in the muscles of the shoulder, which would thus assume the scapulo-humeral type. It may also commence in the lower extremities. The muscles of the face are generally unaffected, but Westphal, Schültze and Grasset have each noted a case of facial paralysis.

*The Skin* is often affected with the condition known as glossy skin, various eruptions, as bullæ, eczema, herpes, are common. The epidermis becomes thickened and horny. There has been described in a certain number of cases (Kahler, 1882) a primitive gangrene of the skin, followed by loss of substance and leaving a whitish puckered cicatrix. Small ulcerations having the characters of perforating ulcers have also been described.

*The nails* are cracked, furrowed, thickened, or may fall off.

The secretion of sweat may be absent in certain regions or it may be exaggerated, in which case the distribution of sweating is very variable. A number of experiments have been made by Grasset, causing the secretion of sweat by injections of pilocarpine, according to the method of Strauss. The appearance of the sweat was delayed, but it was increased in amount.

*The Subcutaneous Cellular Tissue* is in some cases œdematous, in others abscesses or whitlows are present. This latter form of inflammation has been noticed in several cases at an early stage of the disease and supports the idea of identity with Morvan's disease. When we come to the diagnosis we shall have an opportunity of discussing the value of this point. In addition to the œdema, Röth has showed the possibility of the existence of tumours of a doughy consistence in the cellular tissue, being present for a longer or shorter period and then becoming absorbed.

*Vascular disorders* are also of common occurrence. The extremities are frequently cold and cyanosed, and the slightest friction may cause redness which may persist for some time.

The thermometer shows a lowering of the local temperature of about 1° cent.

In other cases there exists an intense redness of the



extremities with a swelling of the fingers, accompanied by an elevation of the temperature. It is doubtless to these disturbances that the curious sensations of coldness or of burning, which the patients feel, are to be attributed.

There may also exist a paralysis of the vaso-motors, so that a slight mechanical irritation of the skin may cause a persistent redness.

*The joints* are sometimes the seat of arthropathies similar to those seen in tabes. This was the case with one of the patients in the Salpêtrière, in which a characteristic affection of the elbow joint was present.

*The bones* are often thickened, they become brittle, and are the seat of exostoses (Déjerine).

Holschewnikoff and Recklinghausen have added acromegaly to the list of trophic troubles which are observed in syringomyelia. It may only be a coincidence in the cases of these authors, for acromegaly appears to be an affection quite distinct from syringomyelia.<sup>1</sup>

*Other Systems.*—The lesions of syringomyelia may extend to the bulb and consequently bring about disorders of deglutition, of the circulation and of respiration. This condition is comparatively rare: usually the digestive and cardio-pulmonary systems are intact. An inequality in the size of the pupils may be observed, caused by the presence of a ciliospinal centre at the level of the cervico-dorsal enlargement of the cord. A unilateral narrowing and straightening of the palpebral slit has also been noticed. Besides the disorders of deglutition in cases in which the bulb becomes affected, there are alterations in the sense of taste (Grasset), and in the sense of hearing (Schültze). To these may also be added intermittent polyuria (Krauss). As to the cerebral portion of the nervous system, it has no share in all the symptoms which we have described, but in some cases there may be nystagmus (Charcot), and amaurosis (Schültze), in others there may be an appreciable degree of mental debility. But neurasthenia or hysteria, which may supervene in this as in other neuropathies, would figure only as associated

<sup>1</sup> Souza-Leite, *Sur l'Acromégalie. Thèse de Paris, 14 Mars, 1890 (Delahaye et Lecrosnier, édit.).*



symptoms and could not be reckoned as an active principle of syringomyelia. The generative sense is usually unaffected. According to our observations it is different with the urinary function. There is no sphincter trouble properly speaking, but we may often see evidences of cystitis which appear to be due to an interference with the nutrition of the bladder rather than to disturbances of the vesico-spinal centre. One of our patients died in this way from a spontaneous perforation of the bladder.<sup>1</sup> M. Röth mentions several cases of disturbances of micturition.

(iii.) The explanation of these symptoms, based on pathological anatomy, is still under consideration, at least as regards some of them. As to the muscular atrophy it is well known that it depends on lesions of the grey matter of the anterior horns.

The sensory troubles may be explained by referring to the experiments of Schiff, which prove that the fibres for conducting tactile impressions are contained in the posterior columns and these being intact, the sense of touch is preserved. The hypothesis was held that the conduction of the other forms of sensibility to pain and to temperature, proceed by the posterior horns, and that alterations in them resulted in the abolition of these perceptions. Nevertheless, M. Röth, having noticed in certain cases that the functional alterations did not correspond with the lesion and, moreover, that it is almost impossible that the pathological process, extending throughout the spinal cord, should be confined entirely to the isolated temperature-conducting fibres, advances the following hypothesis. He admits that the conducting tracts are not differentiated anatomically by their position, but physiologically by their reactions ; that is to say, that being invaded by a new growth, the nerve fibres are not all equally affected. In this case the intensity and the kind of alteration, by their amount, would determine the dissociation of sensibility. For example, an agent, less intense, would only affect the thermal sensibility, leaving the sensibility to touch and pain ; or again, degeneration would be the chemical cause of the

<sup>1</sup> P. Blocq. *Un cas de Syringomyélie. Bulletin de la Société Anatomique Février, 1887.*

abolition of the thermal sensibility, compression due to the neoplasm, the mechanical cause of the cessation of the conduction of painful impressions. The dissociation would, in fact, depend on the varying resistance to the conduction of different impulses in the same nerve, or in the same group of nerves; according as one supposes that a single nerve conducts both thermal and tactile impressions, or that each nerve has a separate function.

Herzen<sup>1</sup> has put forward another theory; he believes the thermal sense to be of two kinds; on the one hand tactile and cold, on the other heat and pain, which would be associated in the cord. The former group (tactile and cold) to be contained in the posterior columns, the latter (pain and heat) in the grey matter. M. Déjerine thinks that it is necessary now to take into account in the pathological physiology, the alterations in the cutaneous nerves, which he has established, as they are the conducting paths for impressions of pain and temperature, but the frequent occurrence of neuritis, now-a-days, in the great majority of nervous affections, does much to diminish the clinical value of such lesions. The trophic changes are due to alterations in the central part of the grey matter of the cord. Starr<sup>2</sup> believes that the trophic centres for the bones lie in the anterior part of the commissure, and the trophic centres for the skin, nails, bladder, as well as the vaso-motor centres in the posterior part.

*The Spasmodic or Tabetic phenomena* seen in the lower limbs are very easily explained by the continued effect of the chief lesion, it may be on the lateral tracts in the first instance, or the posterior in the second, where as we have seen, secondary sclerosis are developed. The scoliosis, so frequently seen in syringomyelia, is more difficult to explain. Is it due to a trophic change in the vertebræ themselves, or, as M. Röth maintains, to a partial atrophy of the spinal muscles? The question is far from being settled and one can only compare this deformity with the condition occurring in another disease due to defective evolution, namely Friedreich's disease.

<sup>1</sup> Herzen. *Pflueger's Archiv*, Vol. 38.

<sup>2</sup> Starr. *American Jour. of Neurol.*

(iv.) The different forms of syringomyelia vary greatly, and I took care to point out at the beginning when I described a mean type, that I do not pretend to describe every form. Nevertheless, from several cases I have examined and from observations I have studied, I think it may be stated that there are two principal forms of this condition, besides the mean type. But I ought first to mention cases in which there was the opportunity of verifying the characteristic lesions by a *post-mortem* examination, and where no symptoms were manifested during life. M. Brühl explains these latent forms by their occurrence in young patients, in whom the symptoms have doubtless not had time to show themselves.

As to the forms which we think it is possible to distinguish, they correspond with the localisation of the neoplasm. In the one form, it commences by an atrophy of the muscles supplied by the ulnar nerve; the other begins by an atrophy of the muscles supplied by the radial. The former is accompanied by spasmodic phenomena in the lower limbs, the latter by tabetic symptoms in the same limbs.

Now in the cervical enlargement, which, as we have seen, is usually the original seat of the glioma, the centre for flexion of the upper extremities is situated peripherally in relation to the centre for extension. Consequently, if the centre for the ulnar in the cord is attacked, the lesion would be observed in the nearest white columns, that is, the lateral columns; but if the radial centre be affected the neighbouring white matter, namely the posterior columns, would become secondarily sclerosed. Thus there may exist three principal types, at least at the commencement; the first, which we have described, characterised by an indefinite invasion of the muscles of the hand—*griffe* Aran-Duchenne—and various affections of the lower extremities, sometimes exaggeration of the patella reflexes, at others a diminution; the second, *cubito-spasmodique*, characterised by an atrophy of the muscles of the hypothenar eminence—*griffe d'extension*—with exaggeration of the patella reflexes; and the third, *radio-tabetic*, characterised by atrophy of the muscles supplied by the radial—*griffe de*

*flexion*—with diminution or loss of the patella reflexes. The special affections of sensibility are common to all three forms. This distinction is, however, only relative, for the extension of the lesion is not as a rule slow in manifesting itself clinically by the greatest variety of symptoms.

## VII.

*Course. Duration. Termination.*—Syringomyelia always commences insidiously and it is generally well advanced before the physician can be sure of the symptoms. Sometimes the trophic changes, consisting it may be of a succession of whitlows, or of erosions or indolent ulcers which show no tendency to heal, begin the attack without attracting much attention, sometimes alterations in the sensibility, which give rise to strange and curious conditions; but as the patient is rarely aware of them, it is only by subsequent enquiries that these early circumstances become known. Often, then, the patient, questioned as to the cause of the numerous cicatrices which he has, mentions different times at which he has been burnt and has only become acquainted of the fact by the blisters it has caused.

In many cases motor disturbances occur first, and it is as well to know that the functional weakness of the muscles is often the first warning the patient may have. The disease subsequently progresses very slowly, more so than any other chronic affection of the cord. The course of the malady is varied by exacerbations which disturb its regularity, and the symptoms may become exaggerated suddenly. A kind of jaundice may occur with complete cervical paraplegia, which gradually disappears, leaving, however, some traces. This circumstance may depend on the possible complication of hæmorrhagic infiltrations which have been noticed in the evolution of the lesion. It may be only from one of these attacks that the patient becomes aware that he is a victim to disease, as, owing to its insidiousness, he may have previously been ignorant of the fact. The patient may describe the commencement of the attack as quite sudden and thus may

put the physician off his guard, if he be not on the look out for such a possibility. In other cases there may be a remission of longer or shorter duration.

It usually terminates fatally. Death may be due to secondary complications, as gangrene, cystitis, perforation of the bladder, or it may occur from the disease itself. Bulbar complications may supervene; it may be that only a nervous cachexia is produced; mortification may set in and the patient die of exhaustion, or more frequently from accidental infections.

Impairment of nutrition is not only a predisposing cause, but at the same time diminishes the power of resisting infection, hence its frequency and extremely powerful effect. Small-pox, typhoid or tuberculosis generally carry off the patient.

### VIII.

*Prognosis.*—Taking into consideration the remarks that have been made about the possible latency of syringomyelia, and knowing that, as M. Röth affirms, one may even hope for recovery, and being aware that life may be much prolonged, even at any stage of the disease, a somewhat hopeful though guarded prognosis may be given. Without being less serious, it is not so fatal as many other myelopathies.

### IX.

*Diagnosis.*—A diagnosis of syringomyelia is quite possible and becomes easier in proportion as the disease becomes better known. There is no symptom which may be said to be pathognomonic; it is only by an appreciation of the symptoms as a whole that the question can be decided. If there be a decided predominance of one of the manifestations, there is a possibility of confusion, accordingly we may classify the differential diagnosis in the following way:—

Cases in which there is a predominant system.

(i.) Trophic disorders	{ Scleroderma, Leprosy, Morvan's disease, Neuritis.
(ii.) Muscular atrophy	{ Cervical hypertrophic pachymeningitis, Lateral amyotrophic sclerosis, Primitive myopathies, Progressive muscular atrophy.
(iii.) Motor disturbances of the lower limbs.	{ Transverse myelitis, Disseminated sclerosis, Tabes.
(iv.) Alterations in the sensibility.	{ Alcoholic paralysis, Hysteria.

(i.) In *scleroderma* or, better still, in *sclerodactylitis*, the symmetrical alterations in the skin of the fingers, the ulcerations which so frequently appear, the sensations of cold which the patients experience, accompanied by muscular atrophy which is sometimes seen in the upper extremities, might suggest syringomyelia; but they are easily distinguished by the preservation of the sensibility and by the peculiar and preponderating character of the inflammation of the skin.

*Lepra anæsthetica* gives rise to symptoms still more analogous; the marked alterations of sensibility and the muscular atrophy which usually assumes the appearance of the Duchenne-Aran type. M. Leloir has reported cases in which the diagnosis was extremely difficult.

In the case of lepra, however, the anæsthesia has a peculiar distribution, very different from that of syringomyelia. There are little islets surrounded by a reddish line, somewhat raised and very sinuous, which may be likened to the appearance of a division on a geographical map. Moreover, the anæsthesia is complete in the majority of cases, and therefore affects the tactile sense. The special etiological considerations, too, of leprosy would be a useful guide. Several affections comprised in the somewhat ill-defined

group named peripheral polyneuritis, give rise to motor and sensory troubles, more or less like those of syringomyelia. The troubles are localised according to the nerves affected; but in the gliomatous condition, at the commencement at least, if the lesion be still localised in the cord, there might be a like distribution of the symptoms. Nevertheless, the rapid course of the neuritis, and, above all, the abolition of the tactile sense which follows, suffices to distinguish them from syringomyelia.

Some writers consider that the disease described by Morvan (de Lannilis) should come within the same nosographical category as syringomyelia. Painless whitlows may be present as a leading symptom, also frequently-recurring phlegmonous dactylitis, which shows no tendency to heal, paresis and analgesia of the upper extremities, and sometimes there may even be scoliosis. In spite of the similarity of these symptoms, they are not the same disease, as a recent autopsy has proved, and it is quite possible to distinguish them clinically. In Morvan's disease the tactile sense nearly always disappears with the other forms of sensibility; the trophic changes predominate, and almost exclusively consist of multiple whitlows, deep cracks and fissures in the skin, and arthropathies of the smaller joints; moreover, in certain cases these affections are symmetrical on both hands and feet, and do not attack the remainder of the body. Finally, the muscular atrophy is less marked, and is not, as a rule, progressive. However, it is possible that the two diseases have been confounded, but still the majority of M. Morvan's observations clearly indicate it as a distinct malady.<sup>1</sup>

(ii.) *Cervical hypertrophic pachymeningitis* gives rise to atrophic paraplegia of the neck, and consequent deformities; while spasmodic paraplegia of the lower limbs may well suggest syringomyelia. But the progress of the meningitis is more rapid, and is accompanied by rigidity of the neck and marked painful symptoms. Lastly, the affections of the upper extremities have none of the complications of analgesia or thermo-anæsthesia.

<sup>1</sup> Morvan. *Gazette Hebdomadaire de Médecine et de Chirurgie*, 1889.



If the ordinarily short course of *lateral amyotrophic sclerosis* be unusually protracted, the muscular atrophy in the upper limbs might give rise to doubt as to the diagnosis; but this may be avoided by remembering that there are no alterations of sensibility in the progress of Charcot's<sup>1</sup> disease, and also that there is a marked exaggeration of the tendon reflexes.

In muscular atrophy of the Aran-Duchenne type sensibility is unaffected. This may also be said of the various forms of primitive myopathies. Besides, in the latter case there are no fibrillary twitchings, and the topographical distribution of the muscular atrophies is characteristically different from that of syringomyelia. We will examine later the difficulties that may result from the combination of primitive myopathy and hysteria.

(iii.) The different forms of non-systematic myelitis, diffuse and transverse, will not long allow of any mistake being made, owing to their course, localisation, and the predominance of motor and sphincter troubles.

*Disseminated sclerosis*, the manifestations of which are very capricious, does not usually affect the sensibility, and has special symptoms, as difficulty of speech, tremors, nystagmus, &c., by which it may be recognised.

It is easier to mistake *progressive locomotor ataxia*, in some at least of its various forms, for syringomyelia. One may imagine a case, for example, in which there is thermo-anæsthesia combined with trophic changes. The loss of the patella reflexes, the presence of lightning pains and slight motor inco-ordination, would not be sufficient to dispel all doubts, for, as we have seen, all these symptoms may occur in syringomyelia; but the muscular atrophy, and the distribution of the thermo-anæsthesia in certain regions, would point to the latter affection, while small patches of thermo-anæsthesia of slight intensity, prominent eye-symptoms, and visceral crises are the particular signs of tabes.

(iv.) A difficulty in the diagnosis sometimes exists in cases of alcoholic paralysis in which the sensory disorders are very

<sup>1</sup> Charcot. *De la Maladie de Morvan. Progrès Médical*, 1 Mars, 1890.



marked, and consist in curious sensations of cold, anæsthesia and thermo-anæsthesia. It is very important to be able to distinguish them. The symptoms are, as a rule, confined to the lower extremities, and make their appearance rapidly. Exacerbations may take place. In addition, there is pain on pressure of the muscles, and the muscular paralysis is out of proportion to the amount of muscular atrophy.

M. Charcot<sup>1</sup> has drawn attention to the possibility, not previously taken into account, of the diagnosis being confounded with that of hysteria. It would be very easy to be mistaken in such circumstances as these; the hysterical symptoms by their combination alone may simulate those of syringomyelia, or hysteria, occurring in a patient the subject of muscular atrophy, may superadd sensory trouble analogous to those of the gliomatous affection. The characteristic dissociation of the sensibility and of the trophic changes occur, as is well known (Charcot, Babinski, Pitres, Weir-Mitchell), among the symptoms of hysteria. But this error may undoubtedly be avoided by a careful examination. The more or less sudden onset of these symptoms, their transient nature, their sensitiveness to sensory impressions, the presence, too, of other signs of neurosis, are sufficient to reveal the true nature of the case. It is none the less necessary to be aware of these coincidences, for ignorance of them has undoubtedly led into error, as M. Charcot has shown, and the confusion of these circumstances might lead to consequences particularly vexatious as regards the prognosis.

## X.

*Treatment.*—In consequence of the remissions which have been observed in the course of syringomyelia—remissions so pronounced as to simulate recovery, it is but right to try some form of treatment to endeavour, if possible, to bring about such a remission of the symptoms, or even, perhaps, to arrest the further development of the disease, in accordance with the hope expressed by M. Röth.

<sup>1</sup> Charcot. *Loc. cit.*

In every case symptomatic treatment should not be neglected. The atrophy of the muscles must be combated by electricity; the condition of the bladder must be carefully watched; the skin must be preserved from all chances of injury to which its analgesic state might render it liable, and the inflammatory complications, especially the more serious one of the cellular tissue, must be most carefully treated.

With regard to the therapeutic measures to be directed more specially against the disease itself, suspension should undoubtedly be tried, on account of the very favourable results that have been obtained from it in other chronic myelopathies. Counter-irritation may also be employed, either by the actual cautery or by the application of iodine over the whole length of the spine. Internally, drugs, whose action tends to promote absorption, may be given, as iodine or the alkaline iodides. Tonics, iron, quinine, &c., should be administered, in the hope of remedying the defective nutrition of the patient.<sup>1</sup>

<sup>1</sup> The scope of this work does not allow us to give a complete bibliography of syringomyelia. This information will be found in the thesis by Brühl, which we have quoted. This work gives references to all the memoirs in the literature of this subject, except the latest ones (March, 1890), to which we have drawn attention; one published in this journal in January, 1890, and another by Krelz in the *Semaine Médicale*, February, 1890.—P. B.

## ON DISORDERS OF THE MUSICAL CAPACITY FROM CEREBRAL DISEASE.

BY Ä. KNOBLAUCH, M.D.

*Clinical Assistant at the Asylum for the Insane of the University of Heidelberg.*

THE present paper was originally intended to discuss a case of disease observed in the clinique of Professor Erb, at Heidelberg—a case of motor aphasia, in which the patient retained the faculty of articulating correctly, in singing the whole text of a song. In any attempt to explain such a condition, the mode of production of musical tones by the human voice has to be borne in mind, as well as the arrangement of commissural paths connecting the cortical spheres for mental representation of sounds with the motor centres of phonation. The mode of combination of the tones produced by phonation, with the sounds emitted by the ordinary action of the muscular apparatus of articulation and respiration, has also to be considered. And further, for the sake of completeness, attention has to be given to the perception of musical tone and its symbols, and to the paths from the sense organs to the higher centres. By this method an idea of the centres and conducting paths in the brain which subserve the perception and production of musical tones and their symbols can be obtained, bearing a close analogy, as was to be expected *à priori*, to the corresponding centres and paths connected with human language. Further, an attempt has been made by the help of Lichtheim's diagram, to design a scheme of those musical centres and conducting paths from which we could theoretically derive a number of disorders of the musical capacity. Some observations of those disorders in cases of aphasia confirm the existence of those symptoms which should theoretically be present; but such observations are few in number, and for the most part

incomplete. Such a fact gives rise to the hope that the scheme about to be described will indicate to observers how those rare cases are to be explained, and also direct their attention to those points which should be most carefully observed.

It is quite clear that there will often be great difficulties to surmount in diagnosing the separate forms of disorder of the musical capacity resulting from cerebral disease, but it is to be hoped that as soon as the attention of observers is directed to these disorders, the number of exact observations will increase, correcting and widening our ideas as to musical centres and conducting paths in the brain.

It seems best to set out those opinions in the same order in which they have been formed in endeavouring to explain the case under observation, and with this view it will be well to give first of all the clinical history of the case.

L. S., æt. six, a little girl from Ludwigshafen, was admitted into the Clinical Hospital at Heidelberg on Feb. 8th, 1887. The child, until then quite healthy, and of normal bodily and mental development, and without hereditary predisposition, became ill with scarlatina on 8th November, 1886. This was followed by nephritis, which, however, disappeared early in December. From that time up till the middle of December the urine was free from albumen, and the child was healthy except that she was a little dull of hearing and complained of noises in the ears. On the 21st December she was suddenly seized with general convulsions. The temperature was raised, there was a large quantity of albumen in the urine, and she remained unconscious for five days. During this time all the muscles remained tense and rigid. On the 26th of December consciousness slowly returned, but there remained a condition of right hemiplegia with aphasia. The child could not speak at all at first. Later on she said "Mamma," and apparently repeated a few words. She could sing the song "Weisst Du wie viel Sternlein stehen," &c., but she could not recite the text of the song or speak voluntarily single words of the same.

In course of time she improved; the albumen disappeared, she became quite conscious, she had a good appetite

and was cheerful, but the paralysis remained to a slight extent.

On 8th February, 1887, the child was admitted into the Clinical Hospital at Heidelberg for electrical treatment, as the aphasia continued, and the right arm and leg remained considerably impaired in their function in spite of the administration of iodide of potassium.

*Present condition.*—On admission the patient is found to be a normally developed, slight but strong child, with fresh red cheeks and a lively restless disposition. Mentally, as far as one can judge, she is very well developed. As she is aphasic she has to make herself understood by gestures; spontaneously she only utters "Mamma." She is able to repeat a few words, but very imperfectly. If one commences the song "Weisst Du wie viel Sternlein stehen," she sings it with the right melody in an automatic way, being unable either to continue or to begin afresh when she once stops. *All the words of the text which she is unable to pronounce spontaneously, are while she sings them articulated perfectly.* The comprehension of spoken language is quite normal. The patient has not yet learnt to read or write. The left side of the body is in every respect normal. The right arm is adducted by contraction of the shoulder muscles; the forearm is acutely flexed on the upper arm, and the hand is bent at a right angle to the forearm; the thumb is adducted and extended; the second and third fingers are hyperextended at all the joints; the fourth and fifth fingers are slightly bent at the metacarpo-phalangeal joint but extended at the inter-phalangeal joints. All those forced positions are caused by the elastic tension of the muscles, and can be overcome if one is not afraid of causing some pain. The tendon reactions at the wrist and elbow are not exaggerated. Sensibility seems normal; there is no atrophy, and no vasomotor disturbances, nor is there any athetoid or choreoid movement. The abdominal reflexes are equal on the two sides. The right leg behaves exactly as the left does; it does not look emaciated. There is slight rigidity of the upper thigh muscles. The patellar tendon reaction is slightly increased on the right side. The plantar

reflexes are equal; sensibility is unimpaired, and the gait is natural. The bodily organs seem to be healthy.

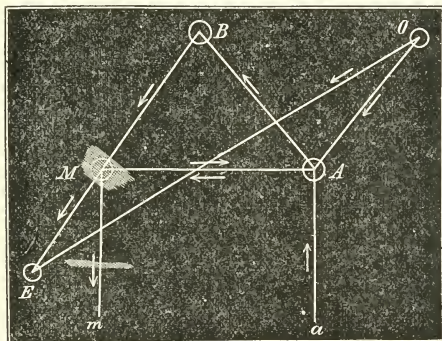
Galvanisation of the head was carried out for some weeks, and the patient was exercised methodically in speaking. Remarkable improvement ensued. On the 21st of February she was able to repeat most words correctly, with considerable trouble, it is true. She could count up to three if some one started her with "one." In the beginning of March she was able to sing the song "Weisst Du wie viel Sternlein stehen" quite alone, and certainly with a much purer intonation than at the beginning of the treatment. On the 8th of March she succeeded for the first time in reciting the text of the song without singing the melody. In the beginning of April the patient had acquired a considerable vocabulary, and she even attempted to form small sentences. In the middle of the same month she could utter almost all words, but could not yet form connected sentences, but she managed to make herself perfectly understood. As the arm had improved considerably the patient was discharged from the hospital on the 13th of April, 1887.

Looking at the acute commencement of the condition in this patient with high fever, loss of consciousness and convulsions, we may conclude that there was present a circumscribed acute inflammation of the brain. Exact localisation is not possible, but the pure motor aphasia indicates a lesion at the foot of the third left frontal convolution, or of the conducting paths which lie just below it, and a lesion of the fibres coming from the left central convolution would account for the hemiplegia. To confirm this diagnosis we have in this case to attend to the speech disorders, comparing them with Lichtheim's diagram.

"The schema is founded upon the phenomena of the acquisition of language by imitation as observed in the child, and upon the reflex arc which this process presupposes. The child becomes possessed by this means, of auditory memories of words (auditory word-representations), as well as of motor memories of co-ordinated movements (motor word-representations). The part of the brain where these memories are fixed may be called respectively the centre of

auditory images, and the centre for motor images. They are designated in the diagram by the letters A and M. The reflex arc consists of an afferent branch *a* A, which transmits the auditory impressions to A, and an efferent branch M *m*, which conducts the impulses from M to the organs of speech. It is completed by the commissure binding together A and M. For comprehension of the meaning of the words a connection must be established between the auditory centre A, and an idea centre B. The next step in the formation of volitional or intelligent speech involves a centrifugal connection between B and M. Our diagram introduces, in consonance with most others, a direct commissure.

FIG. 1.



“Reading requires the existence of visual memories of letters and of groups of letters, *i.e.*, words. We may learn to understand writing through the connection between such visual representations (centre O) and auditory representations. By spelling aloud we bring the auditory centre into action, and thus establish a connection through the path O A between O and B; in reading aloud the tract O A M *m* is made use of.

“The problem with reference to writing is more complicated. The necessary movements have to be learnt and



associated with the visual representations. This is done through the commissure O E, E being the centre from which the organs of writing are innervated. It is more difficult to determine the path through which volitional or intelligent writing is executed. This tract must unite B with E, and clinical facts leave no doubt that it passes through M, but it is doubtful whether it leads directly hence to E, or passes round through A on its way thither."<sup>1</sup>

In the above described case of disease we were able to examine:—

1. Understanding of spoken words (path a A B) intact.
2. Volitional speech (path B M m) lost.
3. Repetition of words (path a A M m) lost.

As the child was unable to read and write we could not examine:—

4. Understanding of written words (path O A B).
5. Volitional writing (path B M E).
6. Reading aloud (path O A M m).
7. Writing to dictation (path a A M E).
8. Faculty of copying (path O E).

The observed symptoms result either from a lesion in M, the centre of motor representations of words or motor centre of speech, or from an interruption of the path M m. We can with certainty exclude a lesion in A, the auditory of speech and an interruption of the conducting paths A M, B M, A B, and a A.

Besides, the following points in the musical sphere were found intact, viz., the correct singing of certain melodies, and the capacity of correctly articulating in singing the words of the song "Weisst Du wie viel Sternlein," &c.

The observation that aphasic patients have retained the faculty of singing correctly, and articulating the words in singing the respective melodies, is not a solitary one. On the contrary, there are numerous observations of the same fact. Thus Falret<sup>2</sup> calls attention to the fact that aphasics may retain the ability to sing. Patients who are able to do so he divides into two classes. Those in one class, while

<sup>1</sup> Lichtheim, BRAIN, vol. vii., 1885.

<sup>2</sup> *Diction., Encycl. des Sciences Med.*, t. v., p. 620, Art. "Aphasie."



still able to sing melodies by the aid of a few sounds, words, or syllables have lost what is strictly language, while those in the other class are able to sing the text of a melody with well articulated words, although they cannot recite the text words of the melody or pronounce voluntarily single words in it, or even repeat them.

To the first series belongs one of Béhier's<sup>1</sup> observations. The patient was aphasic, could only utter the syllable "tan," but yet was able to sing the "Marseillaise" and the "Parisienne," repeating only the syllable "tan" instead of the text. One of Charcot's<sup>2</sup> patients sang the melody of the "Marseillaise" with only one guttural sound, the only one he could utter. Dr. Bouillaud<sup>3</sup> observed one aphasic who was able to sing a song which the patient himself had composed and written in musical manuscript during his illness, and to which he could even play his own accompaniment.

To the second category belong the cases described by Grasset,<sup>4</sup> Hallopeau,<sup>5</sup> Brown-Séquard,<sup>6</sup> Bernard,<sup>7</sup> Gowers, and others, as well as the case just described. An officer who could only utter "*pardi*" and "*b . . .*," and was quite unable to pronounce the words "*enfant*" and "*patrie*," could nevertheless sing correctly, both as to words and music, the first verse of the "Marseillaise." An old piano-teacher suffering from incomplete word blindness and aphasia, could, as a rule, speak only with difficulty, but she was able to sing without difficulty and quite correctly the words of the aria, "La dame blanche vous regarde." Dr. Gowers' case was that of a patient who, from the occurrence of an apoplectic seizure up to the time of his death, was able to speak only the words "yes" and "no." Once when the resident physician wished him "good morning" he replied "ning." On one occasion when another patient in the ward was sing-

<sup>1</sup> Falret, *Loc. cit.*

<sup>2</sup> Bernard, *De l'aphasie et de ses diverses formes*, Thèse de Paris, 1885, p. 195.

<sup>3</sup> *Bull., Acad. imp. de Médecine*, 1865.

<sup>4</sup> Grasset, *Montp. Medical*, 1848, t. xl.

<sup>5</sup> *Traité Élémentaire de pathologie générale*, 1844, p. 578.

<sup>6</sup> *Société de biologie*, 19th April, 1884.

<sup>7</sup> Bernard, *Loc. cit.*, p. 120.

ing "I dreamt that I dwelt in marble halls," he joined in, and sang the second verse alone, articulating every word correctly.<sup>1</sup>

Such observations show that aphasic patients under the influence of musical excitement are able to utter words, the pronunciation of which, apart from that excitement, would be impossible—a condition of things analogous to that which, according to Dr. Hughlings Jackson, occurs in idiotic children, not deaf mutes. Of these, he says that they are able in singing to pronounce far more words than they were able to do in speaking.<sup>2</sup>

What explanation is offered of this condition? Falret<sup>3</sup> believes that it is caused by some deficiency in the memory of words (*lésion de la mémoire verbale*), and he draws a comparison between this condition and that of persons who, in their normal state remember the words of a song only in singing them, being unable to remember them when they try to recite the text.

Gowers<sup>4</sup> regards such a repetition of words as a mere automatic process of speaking. He does not regard the pronunciation of the words of a song as language, at least not conscious or intellectual language, as no one has the intention of meaning what he says through the words of a song. At the autopsy on Dr. Gowers' case, an embolus was found blocking up the left middle cerebral artery, and leading to a disturbance of the whole motor speech region of the left hemisphere. In consequence of this he comes to the conclusion that the automatic utterance was effected by the right hemisphere, a conclusion which would tend to confirm Dr. Hughlings Jackson's opinion that such automatic utterances may be effected by the right hemisphere alone.

It cannot be denied that Gowers' theory has in it much probability, but even if it be accepted the question still remains, through which conducting path of the right hemisphere does the singing of words take place, and how, and at

<sup>1</sup> Gowers' *Diagnosis of Diseases of the Brain*, p. 126.

<sup>2</sup> *Lancet*, 1871, September 23rd.

<sup>3</sup> *Loc. cit.*, p. 620.

<sup>4</sup> *Loc. cit.*, p. 126.

what point, is the union of word with musical tone brought about? This last question is important, not only for the explanation of the case which has been described, but also for understanding the whole mechanism of speech. If the motor centre M in Lichtheim's diagram be connected with the articulation centre by the path M m, an explanation is furnished for the mechanism of whispered language, which is produced by means of the muscles of respiration and articulation, the organ of phonation remaining quiescent. In speaking aloud, just as in singing words, the laryngeal muscles work in combination with those of respiration and articulation, so that the idea centre must dominate not only the articulation centre but also that for the laryngeal muscles—the phonation centre. This is the last in a series of centre subserving the perception and production of musical tones.

Kussmaul<sup>1</sup> has pointed out the existence of such centres and paths, and has indicated them by the dotted lines in his diagram of the centres and conducting paths of speech. The auditory centre of speech will correspond with the auditory centre for tones; the motor centre of speech with the motor centre for tones; the visual centre for letters with the visual centre for notes, and the motor centre for writing with a similar one for the writing of musical manuscript. These centres will be connected among themselves and with the idea centre, by tracts of fibres, as are the corresponding centres for speech, and like them, they will all lie in the left hemisphere.

For the sake of completeness a motor centre for instrumental music must also be mentioned. This will correspond to the motor centre for the instrumental production of human language. These centres are co-ordinated with the motor centres for writing, and are dependent upon the motor centres for speech and tones.

The integrity of these centres is necessary and sufficient for the perception and production of musical tones, and such a scheme furnishes a complete explanation of the fact that

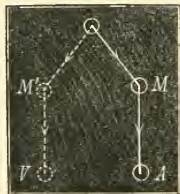
<sup>1</sup> Kussmaul, *Disturbances of Speech*, Ziemssen's *Cyclopædia of the Practice of Medicine*, Vol. xiv., 1878, p. 779.

aphasics sometimes retain the faculty of singing melodies without words. But how does the integrity of those centres explain in motor aphasia the retention of the faculty of singing words, *i.e.*, the production of articulated words as a vehicle for melody? In other words, in what way can we have a stimulation of the articulatory centre after an interruption of the path from the motor centre for speech? There follows naturally on this the wider question, in which way does a stimulus travel, in speaking aloud, from the motor centre of speech to the phonation centre?

In whispered language we have laid down the hypothesis that speech is effected through a path leading from the motor word-centre to the articulation centre. In a similar way the singing of a tone is effected by means of a path leading from the motor centre for tones to the phonation centre.

The following diagram will explain our meaning (fig. 2).

FIG. 2.



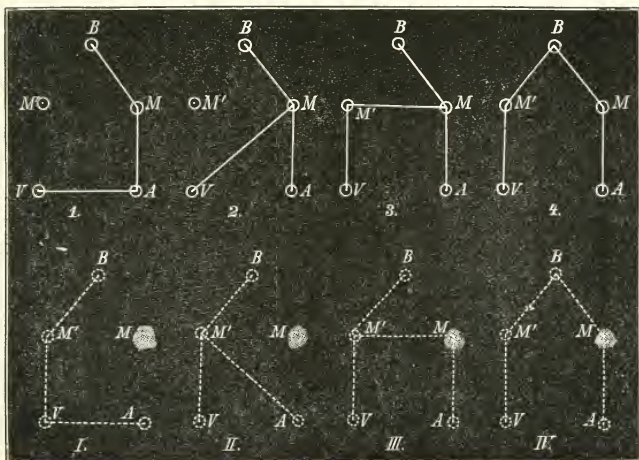
For speaking aloud a connection must be effected between the phonation centre, the articulation centre and the motor word centre; and in singing with articulated words, a similar connection between the phonation centre, the articulation centre and the motor centre for tones. This connection is possible in four different ways as follows:—

In the case described above we may take it upon us to localise the lesion in the centre M, or the conducting path M A. But as there was no inability to articulate while singing, we may exclude from consideration the diagrams iii. and iv., and we may regard the conducting paths in dia-

grams i. and ii. as correct. The articulation centre is thus seen to be connected with the motor centre of tones, either indirectly by the path  $M' V A$ . or directly by  $M' A$ .

The observations which have been mentioned that aphasic patients are not unfrequently able to sing melodies only without being able to articulate the words, make it doubtful whether there is any conducting path from  $V$  to  $A$ , for such a path would not lie at a part of the brain, a lesion of which could bring on aphasic disorders. But if we grant the existence of the path  $M' A$ , we may conclude that the paths  $M' A$  and  $M A$  may be involved in a lesion which would leave the path  $M' V$  intact.

FIG. 3.



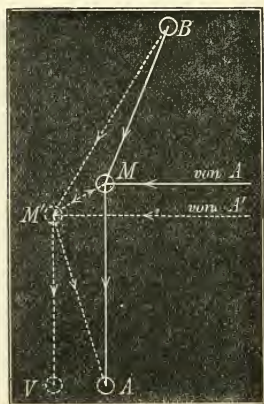
We would, therefore, offer the hypothesis that in singing a melody with articulated words, the articulation centre need not be connected with the motor centre for speech, but with the motor centre for tones through the direct conducting path  $M' A$ . Similarly the phonation centre in speaking

aloud is connected with the motor centre for speech. This view furnishes a complete explanation of the case under consideration.

If this explanation be correct the question is raised, whether an aphasic patient should not be able to articulate any kind of words as the verbal accompaniment to one of his favourite melodies?

Before attempting an answer to this question we shall first try to explain how the words of a song are sung with the melody. In singing a melody with its verbal accompaniment, motor word-representations are produced by the auditory centre for speech, and motor tone representations

FIG. 4.



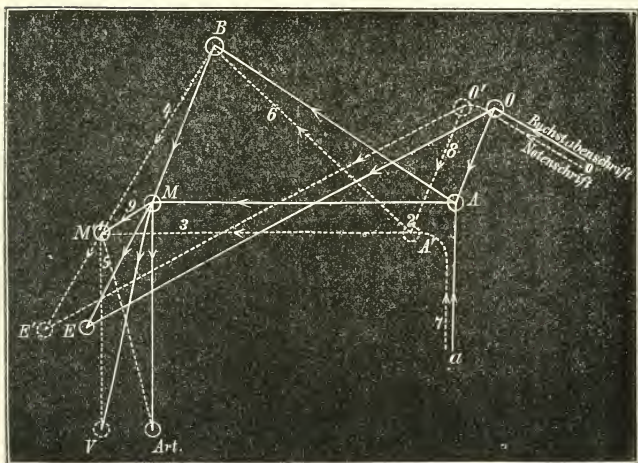
by the auditory centre for tones. The articulation centre being stimulated from the motor centre for tones in singing a melody with words, motor word-representations are produced in the motor centre for tones, *i.e.*, the conducting path MM' must be established. In this path the motor representations of the words of a song are intimately associated with the motor representations of its melody and *vice versa*.



If this path  $MM'$  be intact it must be possible that—

1. So long as the paths  $B M$  and  $M'V$  are intact one can with each melody—produced by volitional reproduction of motor-tone representations, path  $B M'$ —voluntarily associate, by the path  $B M M'$ , and articulate, by the path  $M'A$ , any kind of text words. It must further be possible that—
  2. So long as the path  $A M$  remains intact, the words of the text can be repeated.
- (a) In repeating a melody which was sung with words.  
 (b) In singing notes below which the words of the text were written.

FIG. 5.



Should our view of this path be correct, it will be of value in deciding whether in a given case, the aphasia is due to a lesion of the speech centre or of the conducting path. In aphasia produced by interruption of the path  $B M$ —inner commissural aphasia—what we have described above as No. 1 must be lost; while No. 2, (a) and (b) will be re-

tained; in aphasia produced by lesion of the centre M—central aphasia—Nos. 1, 2 (*a*) and (*b*) are lost; in aphasia produced by interruption of the path M A—outer commissural aphasia, Nos. 1, 2*a* and 2*b* remain.

We shall now proceed to draw the musical centres and paths in Lichtheim's diagram.

In looking at this diagram one can easily read off nine different interruptions of the supposed conducting paths, and their respective symptoms are easily deduced. These nine different forms of disorder of the musical capacity result from interruption partly of the perceptive and partly of the productive paths, so we can divide them into sensory and motor disorders. To name the sensory disorders it will be best to choose the terms "*tone deafness*" and "*note-blindness*," which correspond to the expressions "*word-deafness*" and "*word-blindness*." To name the motor disorders which correspond to aphasia we would propose the term "*amusia*." Steinthal uses the word ἄμουσοι<sup>1</sup> to designate individuals who have lost both words and notes.<sup>2</sup> The Greek word ἡ ἀμουσία means "inexperience, especially as regards music," and without too great violation of language it may be used to mean "disorder of the faculty of musical expression."

We shall now bring forward those nine different forms of disorders of the musical capacity, and describe some of them.

I. Lesion of the centre M'—the motor centre for tones, gives rise to the following symptoms:—

Loss of

- (*a*) Volitional singing, B (M')<sup>v</sup> Art.
- (*b*) Repetition of tones or melodies a A' (M')<sup>v</sup> Art.
- (*c*) Singing of notes, o O' A' (M')<sup>v</sup> Art.
- (*d*) Volitional writing of notes, B (M') E' ;
- (*e*) Writing of notes after hearing a melody, a A' (M') E'.

<sup>1</sup> ἄμουσος musis non initiatus, musicorum modulorum imperitus; ἡ ἀμουσία imperitia, proprie quidem musicorum modulorum et poetices; sed generaliter interdum pro quavis imperitia capitur. Thesaurus Graecae Linguae, ab Henrico Stephano constructus, p. altera. p. 151. Parisiis 1831—1856.

<sup>2</sup> Kussmaul, *Loc. cit.*



## Retention of

- (f) Understanding of tones a A' B ;
- (g) Understanding of written notes o O' A' B ;
- (h) Copying of notes o O' E'.

In this case of "*central amusia*" the whole faculty of volitional singing is suspended, and not only the faculty of singing correctly, but also the ability to sing at all.

## II. A lesion in A'—the auditory centre for tones—gives rise to the following symptoms:—

## Loss of

- (a) Understanding of tones a (A') B.
- (b) Understanding of written notes o O' (A') B.
- (c) Repetition of tones or melodies a (A') M' v Art.
- (d) Writing of notes after hearing a melody a (A') M' E' ;
- (e) Singing of notes o O' (A') M' v Art.

## Retention of

- (f) Volitional writing of notes B M' E' ;
- (g) Copying of notes o O' E' ;
- (h) Volitional singing B M' v Art.

In the analogous speech disorder, voluntary speech is retained, but there is considerable disturbance of the nature of what is called "*paraphasia*." Lichtheim accepts Wernicke's interpretation of this disturbance resulting from a lesion of the auditory centre for speech, the tract for voluntary speech remaining intact. Wernicke assumes<sup>1</sup> "that the nervous influx descending along the path B M m (Fig. 1) sends a branch current to A, and that this *subconscious* innervation of the auditory memories of words secures the correct choice and expression of them, and that irregularities occur as soon as the co-operation of these elements ceases to take place." Lichtheim, however, modifies this view in supposing "that the mere excitation of the auditory representation is not sufficient to secure correct speech, but that this representation must enter into relationship with the concept: that

<sup>1</sup> Lichtheim, *BRAIN*, Vol. vii., 1885, pp. 439, 440, and Wernicke, *Der aphasische Symptomen complex*, Breslau, 1874, p. 23.

therefore the commissure A B must necessarily be intact for the same purpose." Paraphasia, therefore, will result according to Lichtheim, when in the arc B M A B an interruption has occurred in such a way as not entirely to arrest language.

It is probable that in a lesion of the auditory centre for tones there will be disorder of singing similar to what occurs of speech in a lesion of the auditory centre for words. Such disorder will consist in discordant tones and false intervals, the faculty of singing remaining for the most part intact. These musical disorders we name "paramusia." "Paragraphia," as regards the writing of notes, will also, perhaps, take place as a result of a lesion of the auditory centre for tones.

III. Interruption of the conducting path from the auditory to the motor centre for tones path, A' E' is of special interest, as the symptoms, which we conclude from the diagram to be present, agree with those observed by Kast. There are preserved the following:—

- (a) Understanding of tones a A' B;
- (b) Understanding of written notes o O' A' B;
- (c) Copying of notes o O' E'.

The appearance of paramusia shows in accordance with what has been said,

- (d) Volitional singing B' M' v Art;
- (e) Repetition of tones or melodies, a A' B M' v Art  
[a (A' M') v art] ;
- (f) Singing of notes, o O' A' B M' v art: [o O' (A' M') v Art].

The appearance of paragraphia shews,

- (g) Volitional writing of notes B M' E' ;
- (h) Writing of notes after having heard a melody,  
a A' B M' E' [a (A' M') E']<sup>1</sup>

In this case the faculty of singing remains generally intact, but the patient is not able to sing correctly, although he can recognise the incorrectness of the tones which he uses.

<sup>1</sup> Lichtheim (*loc. cit.*, p. 442) draws attention to the fact that in the analogous speech disorders "the path generally used in these acts—from A to M—is interrupted, and that notwithstanding they are not completely in abeyance, because the path A B M may be substituted for it. These actions, therefore,

Kast<sup>1</sup> had the opportunity of observing this association of symptoms in the case of an aphasic patient, who was before his illness "a prominent member" of a musical society in his native place. He describes the case as follows:—

"As a matter of fact the way in which the patient performed the first exercises which I gave him to sing or repeat, a simple melody (*Heil Dir im Siegeskranz*), showed clearly that his musical capacity, at least at present, was far from being such as would satisfy even the modest claims of a rural musical society. I did not fare any better in trying him with hymns, even the most familiar and simple. Yet he always showed that he could get the rhythm of the correct melody; and he also knew the value of every note, yet he sang throughout false tones, and with false intervals, and that in spite of the fact that he was evidently conscious of his defective musical capacity, and consequently not easily induced to lend himself to further experiments. An attempt was now made to get him to repeat tones, and here too, considerable disturbance was apparent, although he recognised the falseness of the tones which he produced and manifested considerable anger thereat. When, however, the patient was got to give me tones which I had to reproduce he remarked the smallest error, corrected me with a loud "Nein, nein," and was only satisfied when the correct sound was given. He recognised all the melodies and songs which were sung to him, and expressed his dissatisfaction if songs he knew were sung incorrectly.

"In the knowledge of writing notes the patient was not sufficiently trained to stand a thorough examination, nor was he able to play any musical instrument."

Kast describes this case as "Broca's aphasia." The are not those of repeating, reading and writing to dictation properly so-called, but are the result of impulses from the 'concept centre,' hence they manifest the same disturbances as volitional speech." In an analogous way one could, in corresponding disorders of the musical capacity, use the path A' B M' for the repetition of tones or melodies, the singing of notes and the writing of notes after hearing a melody, instead of the path A' M'. But in such a case the retention of those faculties is only apparent, and consequently evidences of paramusia and paragraphia will be manifest.

<sup>1</sup> "Ueber Störungen des Gesangs und des musikalischen Gehörs bei Aphasischen."—*Munch. Med. Wochenschr.*, 1885, No. 44, p. 62.



capacity is limited only in so far that it is impossible for the affected individual to articulate the words of a melody in singing. To this group belong the cases already mentioned of Behier, Charcot, Bouillaud.

The symptoms resulting from an interruption of the path M' V, are as follows :—

Loss of

- (a) Volitional singing, B (M'v) Art ;
- (b) Repetition of tones or melodies, a A' (M' v) Art :
- (c) Singing of notes, o O' A' (M'v) Art.

Preservation of

- (d) Understanding of tones, a A' B ;
- (e) Understanding of written notes, o O' A' B ;
- (f) Copying of notes, o O' E' ;
- (g) Volitional writing of notes, B M' E' ;
- (h) Writing of notes after having heard a melody,  
a A' M' E'.

One of Proust's<sup>1</sup> cases, which is unfortunately not very completely described, had symptoms corresponding to those just enumerated. The patient was a lady with aphasia, who had been very musical before her illness came on. She could read and write notes, even compose and recognise melodies sung to her, but she herself was unable to sing the melody.<sup>2</sup>

VI. The interruption of the path A' B would give rise to the following symptoms :—

Loss of

- (a) Understanding of tones, a (A' B) ;
- (b) Understanding of written notes, o O' (A' B).

Preservation of

- (c) Volitional singing B M'v Art., with the appearance of paramusia ;
- (d) Volitional writing of notes, B M' E', with the appearance of paragraphia ;

<sup>1</sup> Proust, "De l'Aphasie," *Arch. Gen. de Med.*, vi. s., t. xix., p. 310, 1872.

<sup>2</sup> Kussmaul (*Loc. cit.*), who also repeats this observation of Proust writes :—"War aber unfähig nach Noten zu Spielen." Proust himself uses the following words about the case :—"Une de mes malades assez bonne musicienne retrouvait parfaitement ses notes, pouvait même écrire de la musique, en composer ; elle reconnaissait un air lorsqu'elle l'entendait, mais elle était incapable de le fredonner."

- (e) Repetition of tones or melodies, a A' M' v Art ;
- (f) Singing of notes, o O' A' M' v Art ;
- (g) Writing of notes after hearing a melody, a A' M' E' ;
- (h) Copying of notes, o O' E'.

VII. If the path a A' should be interrupted there will be loss of

- (a) Understanding of tones (a A') B ;
- (b) Repetition of tones or melodies (a A'), M' v Art ;
- (c) Writing of notes after hearing a melody (a A') M' E'.

The following are intact :—

- (d) Volitional singing, B M' v Art ;
- (e) Volitional writing of notes, a B M' E' ;
- (f) Singing of notes, o O' A' M' v Art ;
- (g) Understanding of written notes, o O' A' B ;
- (h) Copying of notes, o O' E'.

VIII. The interruption of the path O' A' causes “note blindness,” with the following symptoms :—

Loss of

- (a) Understanding of written notes, o (O' A') B ;
- (b) Singing from notes, o (O' A') M' v Art ;

Preservation of

- (c) Volitional writing of notes, B M' E' ;
- (d) Copying of notes, o O' E' ;
- (e) Writing of notes after hearing a melody, a A' M' E' ;
- (f) Understanding of tones, a A' B ;
- (g) Volitional singing, B M' v Art ;
- (h) Repetition of tones or melodies, a A' M' v Art ;

To this category belongs a case related by Finkelnburg<sup>1</sup> of a patient who had lost the understanding of musical notes, but was, nevertheless, able to play well by ear.

What is meant by the term “volitional singing” in the eight forms already described of disorders of the musical capacity resulting from lesions of separate centres, or the interruption of separate paths, is—

1. Singing without words, *i.e.*, the singing of a melody by means of the volitional reproduction of motor tone-representations without the articulation of words—path B M' v :

2. The volitional singing of the words of the text of

<sup>1</sup> Kussmaul, *Loc. cit.*, p. 181.

a song, *i.e.*, the singing of a melody with articulated words effected by the volitional reproduction of motor tone representations, which are already associated with the motor representations of the text-words, path B M' v Art;

3. The volitional substitution of any kind of text to a melody sung voluntarily, *i.e.*, the singing of a melody with articulated words, produced by the volitional reproduction of motor tone-representations, which during the process will get associated with the voluntarily reproduced motor representations of the text words,

$$\text{path B} \begin{array}{c} \text{M} \\ \diagup \quad \diagdown \\ \text{M}' \text{ v Art.} \end{array}$$

By the expressions "repetition of tones or melodies" and "singing from notes" were meant:—

- (1) The singing of the melody, heard or read as the case might be, without words, paths a A' M' V; or o O' A' M' V;
- (2) The singing of the melody with the words of the text, heard or read as the case might be, paths

$$\begin{array}{c} \text{a A M} \\ \text{a A'} \end{array} \begin{array}{c} \diagup \\ \diagdown \end{array} \text{M}' \text{ v Art.}, \& \begin{array}{c} \text{o O A M} \\ \text{o O' A'} \end{array} \begin{array}{c} \diagup \\ \diagdown \end{array} \text{'M}' \text{ v Art}$$

In the symptoms of IX. interruption of the path M M' which are about to be enumerated, we must separate the different forms of volitional singing, the repetition of tones or melodies, and the singing of notes. From the diagram we should conclude that there is loss of

- (a. 3) Volitional substitution of any kind of text to

$$\text{a melody sung voluntarily B} \begin{array}{c} \text{(M)} \\ \diagup \quad \diagdown \\ \text{M}' \text{ v Art;} \end{array}$$

- (b. 2) The singing of the words of a text which have been heard

$$\begin{array}{c} \text{a A (M)} \\ \text{a A'} \end{array} \} \text{M}' \text{ v Art};$$

- (c. 2) The singing of notes with articulation of read words

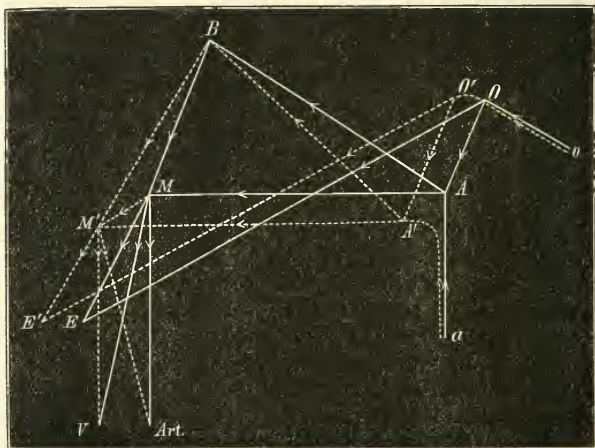
$$\begin{array}{c} \text{o O A (M)} \\ \text{o O' A'} \end{array} \} \text{M}' \text{ v Art};$$



There are preserved,

- (a. 1) Volitional singing of a melody B M' V ;
- (a. 2) Volitional singing of the words of a text B M' v Art ;
- (b. 1) Singing of melodies which have been heard, a A' M' V ;
- (c. 1) The singing of a melody from notes o O' A' M' V ;
- (d) The comprehension of tones a A' B ;
- (e) The comprehension of written notes o O' A' B ;
- (f) The volitional writing of notes B M' E' ;

FIG. 7.



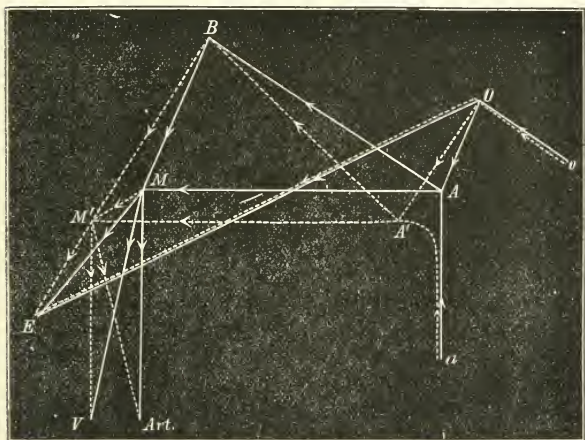
- (g) The copying of notes o O' E' ;
- (h) The writing of notes after hearing a melody, a A' M' E'.

The nine forms of disorder of the musical capacity now described are produced by simple interruptions of the conducting paths. But there can also be a simultaneous interruption of several paths, giving rise to a number of complicated symptoms, as distinguished from the comparatively simple forms which have been enumerated.



We shall not attempt to localize the supposed centres in the brain or to represent the paths leading from and to those centres. It is evident that the auditory centre for tones must be very near the auditory centre for speech, and the motor centre for tones will be very near the motor centre for speech, the former in the superior temporo-sphenoidal convolution, the latter in the third frontal convolution of the left hemisphere. As to the visual centre for notes, and the motor centre for the writing

FIG. 8.



of notes, these, we believe, will be found occupying the same place as the visual centre for letters and the motor centre for the writing of letters. The phonation centre will be situated near the articulation centre, the site of which is, however, still undecided.

Let us now sum up the result of our considerations. For the perception and production of musical tones and their symbols, there are in the brain corresponding centres and paths similar to those connected with the mechanism of speech. From the simple interruption of those paths there result nine different forms of disorder, the localization of

which roughly may be studied by the help of the diagram. To confirm the existence of these centres and paths, it will be necessary, as Kast suggests,<sup>1</sup> "to examine aphasic patients in regard to their faculty of volitional singing, whistling, playing, &c., and their ability to repeat tones or melodies, their judgment of musical tones and scales, their understanding of written notes, and their ability to write melodies from memory and when sung by another," and, further, their faculty of volitional writing and singing of notes.

In examining aphasic patients in regard to their faculty of volitional singing, of repeating melodies or singing notes, it will be necessary to note especially whether the patient can sing only the melody or whether he is able also to articulate the words of the text when singing the melody. Especially must we examine him with a view to ascertain whether he has retained or lost the faculty to adapt any set of words to a voluntarily chosen melody, and whether he can articulate them in singing; and in concluding my paper I may perhaps be allowed to adopt the words with which Lichtheim concludes his work on aphasia. "Though I have ventured the foregoing remarks regarding the probable localisation of aphasic disturbances, I am well aware how limited are the foundations on which we may safely build, and how great a space theoretical reasoning has still to occupy in the discussion. If I have, in spite of this, not hesitated to give expression to my views, it has been on the principle that we must not shrink from the consequences which follow from our hypotheses. In proportion as we draw those conclusions shall we obtain the data by which we must correct, or if necessary, abandon them. Nor can any observer single-handed collect sufficient material to accomplish this task, which requires the co-operation of many, and under those circumstances even erroneous assumptions may not be useless."

<sup>1</sup> *Loc. cit.*, p. 627.

# ON THE PATHOLOGICAL VALUE OF THE GASSERIAN, LENTICULAR, SPINAL AND CARDIAC GANGLIA.

BY W. HALE WHITE, M.D.

*Physician to Guy's Hospital.*

IN the *Medico-Chirurgical Transactions*, vol. lxviii., I gave an account of an examination of a large number of superior cervical and semilunar ganglia, taken from persons dying of various diseases. These ganglia were shown to vary more in size and shape, in different individuals, than any other organ in the body, and these variations were quite irrespective of the age, sex, station of life, or manner of death of the individual. Microscopical examination showed that the number of nerve-cells present in these ganglia was most variable, also that while a few cells had well marked nerve processes and an evident nucleus, very often the nerve-cell was nothing more than a minute mass of bright yellowish red granular pigment. Between these two extremes there were many intermediate forms, but almost all the nerve-cells deviated more or less widely from the typical nerve-cell with a well defined nucleus. The amount of connective tissue was very different in different specimens. For many years past I have been on the outlook for instances in which there was some evidence of pathological change in the ganglia, but except in two patients in whom abdominal malignant growth had invaded the semilunar ganglia, I have not been able to find any which have been affected by pathological conditions.

These results pointed so strongly to the fact that the superior cervical and semilunar ganglia are in adult man functionless organs, that I examined the superior cervical ganglia of a number of mammals other than man, and also

those of a number of human fœtuses. The results were published in the *Journal of Physiology*, vol. viii., No. 2, and I arrived at the following conclusions:—firstly, that human adult superior cervical ganglia vary as much in size as do those of the largest and smallest of other mammals, and that the size of the ganglion in other mammals varies directly as the size of the animal. Secondly, that human superior cervical ganglia exhibit granular pigmented atrophic cells much more frequently than those of other mammals; that this condition, though present to some degree in monkeys, diminishes regularly as we descend in the mammalian scale, till at last it is not seen. Thirdly, that human fœtal superior cervical ganglia do not show any of these changes in their nerve-cells. These facts seemed to me to demonstrate that the superior cervical ganglion is becoming less and less important functionally, the higher we ascend in the animal scale, till in the human adult its minimum of importance is reached. It is, in fact, an atrophied degenerate organ like the coccyx or the appendix cœci.

In *The Journal of Physiology*, vol. x., No. 5, I showed that the pigmented, granular, atrophic, degenerate appearance of the cells could not by any possibility be due to the method of preparation. I also prepared a series of superior cervical ganglia and arranged the descriptions of them according to the age of the patient, and found that of forty-nine adults examined only one showed entirely normal nerve-cells, and of five children examined, all showed entirely normal nerve-cells. I also prepared additional sections of a large number of superior cervical ganglia from animals lower than man, and found that the cells were of the normal type in all save one ape. Preparations were made of a series of semilunar ganglia both from man and the lower mammals. The results agreed so completely with those obtained from an examination of the superior cervical ganglion, that I thought we might conclude that in lower mammals and young human beings the collateral ganglia (if we may judge from the superior cervical and semilunar) are functionally active, but that in monkeys there are evidences of the commencing loss of their function, which

has completely disappeared in the human adult. Sections of the chief nerves attached to the superior cervical and semilunar ganglia showed that they were, both in man and the lower mammals, structurally quite similar to ordinary healthy nerves, and therefore we may conclude that they are functionally active.

The thoracic ganglia, which may be taken as the chief lateral ganglia, were examined in twenty-four persons, and the results were arranged according to the ages of the patients. They proved that the granular atrophic pigmented condition of cell was never so marked as in the collateral ganglia, but some of the cells from elderly persons showed it, while those from children and young adults were free from it. From this the conclusion was drawn that in man the function of the lateral ganglia is maintained well into adult life and only begins to disappear in old age.

Lastly, in the *Guy's Hospital Reports*, vol. xlvi., the attempt was made to see whether these results were confirmed by human pathology, and it was found that they were, for it was shown that when the sympathetic ganglia were destroyed by extensive lesions, such as invasion by growth, no symptoms were produced which could be ascribed to the destruction of the ganglia, and also that in the case of all diseases which have been ascribed in intrinsic lesions of the ganglia, such a pathological explanation will not bear critical examination. Also many instances were brought forward from pathology to show that in man sympathetic nerves possess the same functions as in the lower animals.

Having thus proved that changes in the nerve-cells of the semilunar and superior cervical ganglia in human adults and in the thoracic ganglia in elderly persons are of no pathological importance, I thought it would be advantageous to examine the Gasserian and lenticular ganglia as the most accessible cranial ganglia, the spinal ganglia and the cardiac ganglia. The results of these examinations are given below :—

*Gasserian Ganglia.*—Thirty-six of these were examined. They were taken from persons dying of various diseases.

Some were males, some were females, some were old and some were young. In all cases the nerve-cells were well formed, of the rounded type of nerve-cell, without branching processes. There was a distinct nucleus and a distinct nucleolus. The border of the cell, which took the logwood stain well, was sharp. The capsules, which were well nucleated, were almost entirely filled by the nerve-cells. In many sections one, or perhaps two, nerve-cells showed some granular pigment, but they were never atrophic, and there was never anything like the extreme granular pigmentary atrophic change so often seen in the superior cervical and semilunar ganglia. The nerve fibres were all healthy. The ganglion had a fibrous capsule which sent in fibrous septa. The vessels appeared normal. As has been observed to be the case in the superior cervical and semilunar ganglia, the sections were often crowded with leucocytes. I do not know what significance is to be attached to this condition. In some ganglia a minute capillary hæmorrhage could be seen in the sheath of the ganglion. That it occurred shortly before death was clear from the fact that the blood colouring matter was not absorbed. These hæmorrhages are probably of no pathological value, for it is well known that minute intracranial hæmorrhages are very liable to take place shortly before death. Judging from the condition of the nerve-cells, there can be no doubt that in adult man the Gasserian ganglion is functionally active, and therefore any changes in its cells that may in the future be described, will be of pathological value. Ross (*Diseases of the Nervous System*, vol. i. Sec. edit., pp. 496 and 509) gives references to cases which show that aneurism of the internal carotid has been known to cause intractable neuralgia by pressure on the Gasserian ganglion, and in another patient an exostosis from the petrous portion of the temporal bone produced the same effect in a similar manner. Also Wyss and Kaposi have found neuritis of the Gasserian ganglion in cases of trigeminal herpes. In all these instances, however, there is no reason to doubt but that the pain and the eruption were due to inflammation of the nerve fibres in the ganglion and not to implication of the cells, for both these symptoms may follow an ordinary neuritis.

*Lenticular Ganglion.*—I dissected out from the orbit several lenticular ganglia from the same patients whose Gasserian ganglia I have described, and Mr. E. H. Starling kindly cut ribbons of sections of them for me. These showed that the nerve-cells, although small, are perfectly healthy, each with a distinct nucleus. Many have short processes, others are of the rounded type. The cells fill their capsules. The nerve fibres, both white and grey, are healthy, and there is nothing noteworthy about either the fibrous tissue or the vessels. Changes, therefore, in the cells of this ganglion would be of great significance, and we may conclude that in man it is functionally active.

*Spinal Ganglia.*—I prepared sections from a number of ganglia from the posterior roots of the spinal nerves, taking some from the lumbar and others from the upper cervical region. So many text-books contain histological descriptions of this ganglion, that all I need do is to point out that many granular highly pigmented nerve-cells are to be found, but they are never so extremely atrophic as are those of the superior cervical and semilunar ganglia. The greater number of the cells are, however, very good examples of healthy-looking rounded nerve-cells which stain well with logwood and have a distinct nucleus. Pigmented cells are more common in the lumbar ganglia than in the cervical ganglia. Unless, therefore, the changes in the cells are very gross, it will be necessary, as there is not the same amount of pigmentation of cells in all the ganglia, to contrast a specimen supposed to be diseased with sections of precisely the same ganglion from other persons, before pronouncing its cells to be diseased. Baerensprung long ago described neuritis of these ganglia in a case of herpes zoster, but this was probably only a part of the neuritis of the roots of the spinal nerves which causes this affection. At present we do not know of any pathological affections of the cells of the spinal ganglia, most of which are probably functionally active, these ganglia having only recently begun to become degenerate organs.

*Cardiac Ganglion Cells.*—I cut sections of the wall of the left auricle, near the entrance of the pulmonary veins.



Specimens taken from many patients were prepared, but often the sections did not happen to include any nerve-cells, but they were found in the following cases: a female, aged 73, who died from a strangulated femoral hernia, she had granular kidneys; a female, aged 47, who died from an aortic aneurysm; a female, aged 11 years, who died from exhaustion following hip disease; a male, aged 34, who suffered from a dilated and hypertrophied heart, together with bad kidneys; a male, aged 8 years, who had mitral and aortic disease, together with an adherent pericardium; a male, aged 56, who died from the effects of a cerebral tumour; a female, aged 48, who suffered from atheroma, cerebral hæmorrhage, and mitral and tricuspid stenosis; a male, aged 64, who had chronic Bright's disease; a male, aged 58, who died from general tuberculosis; and a patient whose cause of death was not stated. The nerve-cells were always considerably smaller than the cells of the larger ganglia in the body, but they appeared to be perfectly healthy cells without any pigmentation or granular change.

The conclusion, therefore, to which I am led is, that the cells of the semilunar and superior cervical ganglia of human adults are degenerate and functionless, and changes in them are of no pathological value, but such changes in the thoracic ganglia and the spinal ganglia, which are only partially degenerate and are probably in parts functionally active, may be of some importance; but the cells of the lenticular, Gasserian and cardiac ganglia are in human beings healthy looking and apparently functionally active, so that we may expect that any alterations in them will turn out to be of great pathological interest.

In the central nervous system groups of small strongly pigmented cells are often seen, as for example in the substantia nigra, substantia ferruginea, and smaller groups in the pons. An exact account of the situations of such cells will be found in Dr. Hill's translation of Obersteiner's "Anatomy of the Central Nervous Organs." These small cells are perhaps the degenerate representatives in man of groups of cells which in the lower animals had a functional importance for which in man there is no demand. If so



their existence supports the view here expressed, that many nervous ganglia are in man degenerate, and in reality represent organs which were active in the lower animals.

The ganglion of the trunk of the vagus has been described by Levin (*Lancet*, July 14, 1888, p. 78) as containing granular pigmented cells, probably therefore it is in man an atrophied degenerate organ.

## REMARKS ON MR. SULLY'S PAPER ON THE PSYCHO-PHYSICAL PROCESS IN ATTENTION.<sup>1</sup>

BY PROF. A. BAIN (ABERDEEN).

WHEN we pass from the voluntary control of the muscles in full actuality to the control of the thoughts or ideas, the transition is a marked one. As a great number of our ideas are representations of images containing a muscular factor, it is easy to suppose that the will operates upon the muscular ideas, as it did upon the reality. Indeed this is the only version that we can give of the manipulating, say, of visual pictures. We can survey these in all their parts passing from one part to another, as we did with the original. We can dismiss the entire picture from the present consciousness, just as we turned away from viewing the original. In both cases we are under the domination of motives; given an adequate motive the result follows on the same lines in both cases.

The kind of consciousness that accompanies ideal movement is the consciousness of activity or energy. This is of course slightly different in the cases of the actual and the ideal; the precise difference needs not to be dwelt upon for the present purpose. Enough to say, that it is a species of active or motor consciousness, and can be distinguished from passive or sensory consciousness, although in most cases the two are combined. This consideration is of some importance in the questions that have arisen, as discussed in the foregoing paper. More particularly, in the manipulation of images which by their nature seem exempted from ideal movement, there being still a consciousness of motor or active strain, it has to be seen in what manner this consciousness is embodied.

<sup>1</sup> Read at the Neurological Society's Meeting, December, 1889. Published in *BRAIN*, Summer number of 1890.

The difficulty is given by Mr. Sully in the case of visualising a shade of colour; it is still more prominent in the senses that do not repose upon extension at all, as hearing and smell. A sound has no muscular extension in itself, yet it has a plurality of distinguishable properties—pitch, loudness, sweetness, volume, *timbre*. We can attend to any one of these to the exclusion of the others, and we are aware that we are under the active consciousness; we can pass from attending to the pitch, and attend to the sweetness or *timbre*, and the transition is an active exercise, whether through muscles or otherwise, at all events we are not aware of any difference in consciousness between this transition and the transition in attending to a new portion of our field of view in vision. The same illustration could be pursued with complex tastes or odours where the transition is genuinely active, but not a transition from one point of space to another.

The line of investigation suited to such a problem seems to be this. In the first place we must exhaust all the possibilities of muscular change in the region of ideas. We may be able to discover subtle modes of putting forth muscular energy different from the sweep of the eye over space. It is well known that any ordinary act of attention involves a quantity of muscular expenditure in determining the general attitude of the body—an expenditure both positive and negative, that is, both of action and of inhibition, with all which there is necessarily a muscular consciousness. This would be sufficient to account for the active feeling in the non-extended senses, and in their ideas. What it still leaves is the exertion of passing from one attribute to another, both co-inhering in the same point of extension. As regards hearing in particular, it seems to be the opinion of Münsterberg that an element of extension enters into this sense. Probably he would not say the same of odours or tastes, or even touches where the properties co-inhere, as temperature and hardness, and such would be the cases for studying the muscular strain accompanying a change of attention from one quality to another, as from the intensity to the sweetness of an odour. Something must depend upon the determining

circumstances of the transition. It may be, so to speak, spontaneous, that is, it may be due to exhaustion of the attention upon one quality, and consequent emergence of the other into full consciousness. Here, of course, there would be no apparent muscular act involved, neither would there be any muscular consciousness beyond what is implied in the general attitude, due to the occupation of mind upon the sense. Again, the transition may be due to some motive, as an end or purpose rising in the view, and then there would be something of the nature of an active stimulus, however we may choose to define it. It would come under the class of motives growing up in the course of the thoughts, and in the ultimate analysis would come under the activity that regulates the transition from one thought to another. So far as these thoughts involve pictorial imagery, they fall under the case that we start with as presenting the least difficulty, or rather no difficulty. In so far as not pictorial the real difficulty is repeated.

One suggestion remains. While in the case of extension we can assign actual movements of muscular parts in the case of co-inhering properties, it is conceivable that there may be fictitious movements of a very subtle nature in imitation of the veritable muscular changes. By their very nature such movements may be so subtle as to elude definition. The choice would seem to lie between this supposition and the assuming of a muscular consciousness wholly detached from muscular expenditure actual or ideal, and yet indistinguishable subjectively from the true muscular consciousness. Obviously, this supposition is too improbable to be adopted, until every other resource fails.

BY M. ALFRED FOUILLEE (PARIS).

I. *Psychological point of view.*—(a) The chief question is to find out whether attention is a *complexus* of passive sensations. According to Münsterberg, whenever ideation is voluntary and created by attention, the instant of the clear consciousness of an idea, A, has been preceded by another state of consciousness already containing the idea A. It

would thus merely be the relation of a clear, to an obscure antecedent state of consciousness which makes us believe that the willed and sought idea—emerging from the depths of subconsciousness instead of being born, as sensation, *ex-abrupto* from the outside—comes from ourselves, and is excited by our own activity, our attention. Activity would thus nevertheless be but an illusory principle, and attention be a mere relation between essentially passive representations.

We do not admit Münsterberg's explanation to be adequate; for it is not enough that a representation pre-exist in an obscure form to its clear apprehension to generate in us the feeling of activity. Just as a very distant and feeble light can get nearer and stronger without giving us that feeling, thus a reminiscence, confused at first, can suddenly grow clear without any seeking or effort on our part. Whenever attention is present we have a feeling of mental *work*, of expenditure of energy. Mere intensity of representation is not identical with attention. It is not even sure that attention directly by itself augments the intrinsic intensity of sensations, for it would thus alter them; it rather makes them more distinct, *qualitatively* more differentiated, by isolating and protracting them in consciousness. Without attention they would have been transitory, and fused in the common mass. We must avoid mistaking modifications of quality, duration and relation, for modifications of intensity.

(b) Attention is always called forth by an emotion or an interest, by pleasure or pain. In opposition to Mr. Sully, who calls this the second stage of development of attention, we look upon it as the first, or rather as the very ground of attention, which is a *desire* to know, an appetitive phenomenon. Intensity of a sensation may appear to provoke by itself "reflex attention;" but this is not a primitive, but only a derived, psychological fact. This reflex attention consists always of a desire or a fear, more or less disguised, as for instance when a thunderclap fixes our attention.

(c) Though attention be always an active phenomenon of desire and will, it is not thereby *free*; it is necessarily determined by the interest we take in knowing a thing. Wundt's *apperception* is but attentive perception, and does

not possess any of the characters of an indeterminate and free activity.

II. *Physiological point of view.*—(a) Mr. Sully fully shows, against Prof. Ribot, that the mental attitude of attention does not appear subjectively as identical with *muscular* consciousness; but we do not think him entitled to conclude, as he does, that attention is not a *motor* phenomenon. Every movement is not muscular, as Mr. Sully, Ribot and Münsterberg seem, erroneously, to imply. Attention is a volition to feel and know, not to move muscles. The motor impulse expends itself first upon the sensitive and ideational centres of the brain, and the muscles are involved merely in synergic, or sympathetic, movements. Muscles are moreover but the tardy products of a long evolution—a perfected apparatus of amplification for reactive movements which from the first existed, and still exist, in every living cell. Mr. Sully reminds us, with Helmholtz, that we can attend to an object in the regions of the visual field beyond the point of fixation, and he asks where in this case be the motor factor. We answer that it lies in the liberation of cerebral energy upon the sensory centres of vision, not upon the ocular muscles.

(b) I do not believe in the existence of true specialised motor centres; each centre is simultaneously sensory and motor, because it receives and restitutes motion. Excitation of a cerebral cell determines a passive sensation; motor reaction produces the active feeling of attention.

Münsterberg denies that motor impulse is accompanied by any psychical phenomenon before movements have taken place in the limbs through muscular action. This negation is entirely gratuitous. Everything that produces rupture of cerebral equilibrium must produce a contrast in the *cœnæsthesia*, and contrast is the very condition of distinct consciousness. Now motor discharge, even when it is spent not upon muscles, but intra-cerebrally in the form of innervation of sensory and ideational centres, involves a sudden transformation of tension into *vis viva*, of potential energy into actual energy. This sudden rupture of physical equilibrium must have its psychical counterpart; it consti-

tutes cerebral work of a very different nature to cerebral passivity, and we call this work the substratum of attention. We are not conscious of the centrifugal current once let loose upon the muscles beyond the cerebral centres; we then experience as a sort of *choc en retour*, mere passive muscular feelings. But we are conscious of the *starting* of a centrifugal current at the instant it is liberated in the brain. Attention, in a word, is by no means an essentially *muscular* phenomenon, it is so merely by accident or repercussion, but it is an essentially *motor* one, because it is of the nature of an appetite, or effort, and that there are no appetite or effort without sudden liberation of cerebral energy.

(c) Is the mechanism of attention *excitatory* or *inhibitory*? We think it is both, for the very reason that certain parts of the cerebral cortex are excited, others are inhibited. We do not moreover assume the existence of special inhibitory centres, the phenomenon being most likely but a resultant of nerve currents neutralizing one another.

*Conclusion.*—Ideation always contains an element of appetite, hence in some degree of attention: it is an ideomotor process. The main principle of our theory of “idea-forces” is precisely that there is no idea or representation that does not imply a certain effort, a tendency to realisation, an appetite of which the physical counterpart is motion. An idea existing alone would *ipso facto* realise itself in movements; conflict of ideas is conflict of appetitions and motor tendencies. Attention implies a dominating idea only because it is a dominating on which, asserting itself over all others, gives rise to a direction or adaptation of movements, first in the brain, next and secondarily in the muscles.

Attention has played an important part in natural selection, in protecting the individual against destructive influences and in giving it the means of adapting only not itself to its surroundings, but the surroundings to itself. Hence we conclude that if the psychical element of attention (which is consciousness of activity) were superfluous and a mere useless epiphenomenon, it would never have been developed, for, on evolutionary principles, everything that has

been developed and lasts, owes its persistence to its utility. We think that it is the same process, of which we are subjectively directly cognisant as "will to know" a certain thing, or attention, having as its physical substratum a cerebral movement of innervation ending in muscular movements. So far from being a "superadded aspect," as Maudsley and Ribot put it, its mental elements are the very ground of attention and movements merely its external manifestation. What looks like a mere mechanism, is ultimately reducible to mind.

BY MR. SHADWORTH H. HODGSON.

If a few words may be said by one who is neither a professed neurologist nor a professed psychologist, though deeply interested in both subjects from their close connection with philosophy, I would first express my agreement with Mr. Sully's paper on the advantages which psychology may derive from the systematic study of neural organs and processes, which it is the special object of this Society to pursue. Especially it may be expected to throw light upon the fundamental question of psychology, without a definite hypothesis concerning which no progress can fairly be looked for, I mean the question: What is the agent or agency immediately concerned in sustaining the phenomena of consciousness, and determining their order? Is it a substantive but immaterial agent; or are the phenomena of consciousness, or some of them, real agents in determining their own emergence and their own order; or is it the neuro-cerebral organism? Now we know that the last of the three is a *vera causa* of the emergence of states of consciousness, and of the order in which they occur, in many cases, such as the receiving of sense impressions and the spontaneous association of ideas. But that either of the two former is a *vera causa* of the emergence of consciousness, or of the order of its phenomena, still remains to be proved. For my own part, I find myself unable even to form a definite conception of a substantive immaterial agent. And no proof, so far as I am aware, has ever yet been given, that states of consciousness



are ever self-produced, or that when produced they exercise any influence on the subsequent course which consciousness takes. When, *e.g.*, we say that pleasure or pain prompts us to certain actions, there is always a neural agency supporting the feeling, to which the prompting of the action may be more reasonably attributed than to the feeling itself. But however this question may finally be decided, one thing seems clear—it is that the only legitimate way of approaching it is to distinguish, not between mind and matter, or psychical and physical action—as if these were already known terms—but between consciousness on the one hand, and what I may call the proximate real conditions of consciousness on the other. Next will come the question, What is the nature of that or those real conditions, so distinguished from consciousness taken, in the first instance, simply as a conditionate? It is, I think, first and foremost on this cardinal question of psychology that light may be expected, close and minute study of neural activity.

## Clinical Cases.

### ON A CASE OF ATAXIC PARAPLEGIA WITH AUTOPSY, AND A CASE OF LOCOMOTOR ATAXY; SUSPENSION; DEATH FROM SEPTICÆMIA; AUTOPSY.

BY J. MICHELL CLARKE, M.A., M.B. CAMB., M.R.C.P. LOND.

*Assistant Physician and Pathologist to the Bristol General Hospital.*

JANE P., æt. forty-nine, married; a dressmaker. The patient was admitted into the Bristol General Hospital in July, 1888, under the care of Dr. Skerritt, who has kindly permitted me to publish the case. Her family history contained nothing of importance, except that her father died suddenly from heart disease, and one brother suffers from the same complaint.

She had two children; had always been strong with the exception of an attack of renal dropsy seven years ago, and "inflammation of the bowels" six years ago. She had never had syphilis, and had always been temperate; she had for some time not had sufficient food, and lived in a damp house. The menopause came on at the age of forty-six, till then she had always been perfectly regular.

The present illness began after the menopause, with gradually increasing weakness in the legs which felt "dead and heavy," pains in the back, and giddiness. The notes taken at this time state that she was fairly well nourished and of healthy appearance. There was no anasarca. The lungs were normal. The cardiac impulse heaving, in the fourth interspace in the nipple line, and attended with a systolic thrill: the first sound was re-duplicated at the apex. The liver dulness was normal; she had some difficulty in passing her water, but it is not said that it contained albumen. Vaginal examination detected no abnormality.

Her gait was feeble and hesitating, she walked on the back part of her feet, and the body was thrown backwards as if to prevent herself from falling forwards. The knee-jerk and plantar reflexes were exaggerated; the muscles of the legs soft and flabby; muscular power in arms good. No affection of special senses; vision defective; pupils contracted, but equal and acted well to light and accommodation. There was a little tenderness over the lower lumbar vertebræ. She was discharged from the hospital

better, but again came up as an out-patient, April 10th, 1889, when I first saw her. She then complained of increasing weakness in the legs, and could hardly walk, but could move the legs well when lying on her back. \* For the last nine months the weakness had also affected her arms, so that she could only raise them to her head with difficulty. She feels constantly cold, and is sore all over. She had no pains in the limbs, but suffered from pains in the back, passing round her body. She also complained of shortness of breath, palpitation, dyspepsia, with sickness after food, and giddiness, which was much increased in the dark. She had no numbness or loss of sensation in the legs, but felt sometimes as if "walking on rollers." Muscular twitchings troubled her much. On examination the patient was very anæmic, with a distinctly lemon-coloured tinge of complexion; she was not emaciated. Physical signs as given above, except that a faint systolic murmur was heard over the second right costal cartilage, and at the apex. The urine contained a trace of albumen. Both legs were solidly œdematous, pitting on pressure.

Muscular power very feeble both in arms and legs, the muscles were generally flabby, but there was no wasting nor rigidity, and they reacted normally to the Faradic current. The knee-jerks were much exaggerated, and the tendon-reflexes in the arms brisk. Ankle clonus was not obtained. Plantar and abdominal reflexes normal. Her gait was peculiar; it was feeble, her feet seemed to cling to the ground in starting, but at the same time her movements were ataxic and her feet thrown out uncertainly. She turned round with great difficulty, was unable to stand with her feet close together, and fell at once if she closed her eyes (Romberg's symptom). The movements of the arms were slow and weak, but showed no ataxy.

Sensation everywhere normal to touch and temperature; there was a little general hyperæsthesia to pain. No subjective affection of sensation. There was slight tremor of the head and neck, which she said came on about eighteen months previously; there was also slight tremor of the arms, not increased on movement, very fine in character, and in no way resembling the tremor of disseminated sclerosis. Her speech was slow, rather drawling, with slight blurring of words, and tendency to elision of syllables. There was no trembling of the lips, but the tongue was a little tremulous. No paralysis of any cranial nerve. Pupils equal, acted well to light and accommodation, of medium size. The optic discs were white and atrophic, especially the right; the vessels were large, and had white lines on them; other parts of

fundus normal. Vision very defective, almost reduced to perception of light in the right eye, and the field in the left eye was concentrically contracted to a small area around the fixation point. Intellectual power good, except that lately memory was defective. She was highly emotional. She was admitted under Dr. Skerritt, and the next note of interest is on May 7th, when it is stated that the tint of her skin was a pale primrose colour. The blood from the finger looked pale and watery, and red corpuscles were found to be very small—number not stated—but in normal ratio to white corpuscles. Nystagmus was now occasionally noted. Pulse 100. Temperature 100·4. On May 13th, patient was in a somnolent condition, and she constantly remained in this state till her death a few days later. She died apparently from increasing weakness, the transparent, yellowish appearance of the skin became more marked, but changed to a deeper tinge during the last two days of life.

At the autopsy made sixteen hours after death, the pale yellow colour of the skin noted above was conspicuous; the blood was pale and watery. There was a slight superficial erosion of the skin over the upper part of the left side of the sacrum. The right pleura contained 16 ozs. of serum; the lungs were œdematous, but otherwise healthy. The heart weighed  $11\frac{1}{2}$  ozs.; its walls were flabby and uncontracted; aortic and pulmonary valves competent; mitral and tricuspid incompetent, the former admitting three, the latter five fingers. The mitral curtains were much thickened, especially at their free borders, by firm fibrous tissue. The walls of the left ventricle showed fatty degeneration, especially well-marked in the muscoli papillares; the openings of the coronary arteries were normal. Gall bladder contained a little dark bile; bile ducts patent. Liver on section was somewhat fatty, its substance soft and friable. Spleen normal. Supra-renal capsules appeared normal. Both kidneys were pale, the cortex being large in proportion, pale, yellowish-grey in colour. The capsules stripped easily. Stomach, intestines, bladder and uterus healthy.

Cranial vault and bones at base of the skull healthy; membranes both of brain and spinal cord healthy.

The brain weighed  $41\frac{1}{2}$  ozs.; was small; and there was slight excess of cerebro-spinal fluid, which was of a bright yellowish colour. The brain was injected with Müller's fluid and hardened; sections were then cut, which were normal in all respects. Sections of cortex microscopically examined were healthy.

Sections were made of the nerves of the cauda equina, but nothing abnormal was detected in them.

The spinal cord was preserved in Müller and then for three days in spirit, and the sections stained by Weigert's process.

Sections of pons, medulla, and basal ganglia were prepared in the same manner.

On examining sections of the lower lumbar and upper sacral (fourth and fifth lumbar, first and probably also second sacral) posterior roots, these were found to be partially degenerated; increase of connective tissue with numerous fibres in which the myelin sheaths were breaking up or had totally disappeared. There were numerous oil droplets of varying size scattered over the section. Both large and small fibres showed degeneration, but the healthy large fibres appeared to be much more numerous than the small.

In the upper sacral region (fig. *k*) a small area of degeneration was present in each posterior column—in the position indicated in the figure—most intense next the posterior median fissure; there was also on each side in the position of the crossed pyramidal tract a small wedge-shaped sclerosed area. The rest of the cord was normal; the posterior root zone healthy, and the nerve-fibres from the posterior roots which enter the horn directly, and those which sweep round the inner side of the corpus spongiosum to enter the neck of the horn were numerous and healthy.

At the level of origin of 3rd-4th lumbar nerves (fig. *i*) the degenerated area is larger, occupying about the middle parts of the posterior columns, separated by a narrow zone of undegenerated fine fibres from the posterior median fissure and not extending either to the posterior commissure or to the posterior surface of the cord. The area of degeneration in region of crossed pyramidal tracts is larger. Posterior root zone, fibres entering posterior horn, and all other regions of the cord healthy.

So far the areas of degeneration in the posterior columns were not completely sclerosed; some healthy fibres were seen running in them, more so in the sacral region than higher up, but at about the *second lumbar roots* the sclerosis is nearly absolute, very few healthy fibres running in them. The degenerated region here reaches further towards the commissure, a narrow band of healthy fine fibres, some running longitudinally, some obliquely, still separates it from the posterior median fissure and widens out towards the posterior surface. At level of junction of dorsal and lumbar regions the degenerated tract, which is larger but occupies the same position as in the previous section, now first reaches the posterior surface of the cord. The sclerosis is most dense in its centre; the undegenerated tract bordering the posterior median

fissure is much narrower, but broadens out behind into a wedge-shaped area, which consists almost entirely of fine fibres, running longitudinally.

At tenth dorsal the posterior median columns (columns of Goll) show complete sclerosis, which does not, however, extend quite to posterior commissure, and leaves well-marked the unde-generated wedge-shaped area posteriorly. At eighth dorsal (fig. *g*), this latter, though still prominent, shows distinct signs of sclerosis, at the third dorsal it is inconspicuous, containing only a very few healthy fine fibres, and above this level it totally disappears. From the eighth to third dorsal the degeneration in the column of Goll is most extensive and runs close up to the posterior commissure. Up to the eighth to tenth dorsal nerves, degenerated fibres are found in the posterior roots as they enter the cord; after this point they become few in number, though degenerated fibres appear in posterior roots in sections taken from cord as high as second to third dorsal, but not above this. At the entrance of the posterior root into the cord itself the fibres which pass directly into the tip of the posterior horn, and those which run in the internal root-zone before entering the posterior commissure, are healthy at all levels. In the lower part of the dorsal region in the most posterior part of the postero-lateral columns are to be seen a few degenerated fibres, probably passing through these columns on their way to pass upwards in the columns of Goll; in the lumbar and upper half of dorsal regions, this was not made out. With this small exception the posterior lateral columns and the posterior root-zone (Lissauer's tract) were everywhere normal.

Tracing up the column of Goll the figures show that in the cervical region its posterior end is chiefly affected, that it tapers off anteriorly and at the level of the first cervical nerve forms a wedge-shaped area of degeneration in the posterior third of the column only.

The degeneration of the crossed pyramidal tracts also extends right through the cord, and is comparatively greatest in amount from the lower dorsal to the mid-cervical region. It begins as a wedge-shaped area extending to the surface of the cord lying in front of the posterior horn, and separated from it by a band of healthy tissue; this band is broadest in the sacral and lumbar regions, becomes very much narrower in the dorsal, in the upper part of the latter region the sclerosed area closely approaching the posterior horn, and broadens out again somewhat in the cervical part. At the tenth dorsal there is some indication of degeneration on the margin of the cord in front of the crossed pyramidal tracts,

this becomes plain at the eighth dorsal, and is thence continued up the cord, and in the middle and upper cervical regions this marginal band of degeneration extends on the margin of the cord both anteriorly and posteriorly to the crossed pyramidal tracts. The shading in the figures is meant to indicate that degeneration was absolute at the surface and for some way inwards and then was not quite so intense in the rest of the degenerated tracts.

As to the posterior vesicular columns and cells; in the lumbar and lower dorsal parts of the cord, few or no cells appeared in sections at different levels, and the plexus of fine nerve-fibres was atrophied, while there was excess of connective tissue nuclei and some granular *débris*; at the same time the fibres entering the column, and the longitudinally-running fibres bordering it were perfectly healthy. Exception must be made for some sections though the first lumbar, where from five to six healthy cells could be counted; but here the plexus of fibres was also deficient. At the levels of the ninth to eighth dorsal the cells were more numerous, but with very few exceptions had undergone fatty degeneration, and here also the nerve-fibril plexus was deficient.

A group of cells at the level of the third dorsal which occupied the same position in the cord as the posterior vesicular column, was also degenerated, containing fat-droplets.

It is doubtful whether there is slight degeneration of Türk's column in upper dorsal and lower cervical parts. The rest of the antero-lateral columns, cells of anterior cornua, and anterior roots were normal. At the decussation of the pyramids the position of the degenerated areas is indicated in fig. *b*. The sclerosed layer on the posterior surface at the commencement of the funiculus gracilis is the upward continuation of the degenerated column of Goll: the motor tracts here crossing, show distinct but not complete sclerosis; and an area of degeneration lies between the tubercle of Rolando and the margin of the section, corresponding to the direct lateral cerebellar tract below.

This latter area in a normal section through the same spot is occupied exclusively by large fibres. Conspicuous at this point were large vessels in the central grey matter with thickened walls.

In a section (fig. *a*) of the medulla at about the level of the pneumogastric nucleus and the lower part of the olive, there is a superficial degenerated layer in the grey matter of the floor of the fourth ventricle, and this can be traced gradually disappearing a little higher up.

At the margin and just posterior and external to the



olive is an ill-defined area of partial sclerosis only, somewhat wedge-shaped, the base outwards. It occupies much the same position as the degenerated area at the same level in the next case (of locomotor ataxy) but is not so completely degenerated and therefore not so well defined. At this level the anterior pyramids are perhaps a little degenerated, but above this they appear perfectly normal; so that the degeneration of the motor tracts may be said to stop at or just above the decussation.

The restiform bodies show an increase of connective tissue. Sections of the upper part of the medulla and of the pons showed that they were healthy except in the respects above mentioned. The nuclei of the cranial nerves and the cortex of the motor region was normal.

Careful search was made for islets of sclerosis or other morbid changes, but none was found beyond those described.

To sum up the lesion, the parts affected were that part of Goll's column which carries fibres from the lower limbs, the direct cerebellar tract, and the crossed pyramidal tracts, with atrophy of cells of posterior vesicular column, at any rate for a considerable part of its extent. The lesion would appear to start from the posterior roots in the lumbar and sacral regions, sparing entirely the fibres connected with the posterior horn, and picking out the fibres which pass from the posterior root directly to the cerebellum by the column of Goll. The course of these fibres is well shown; until the lower dorsal region is reached they are separated from posterior median fissure by a tract, which extends to the posterior surface and persists through the greater part of the dorsal region as a wedge-shaped tract, becoming smaller until it finally disappears about the third dorsal. No degeneration was found in the cord below the upper sacral region; this tract may perhaps contain the fibres from the lower sacral region and in that case it would seem that fibres from lower parts of the cord running in the posterior median column in the lumbar region, lie nearer the posterior median fissure than those from parts higher up, and that in the dorsal region the former lie posteriorly to the latter. It is difficult, if this is so, to understand why they should disappear in the upper dorsal region.

On the other hand, this little tract may be commissural, but in any case does not reach the medulla; in what way its fibres are disposed of I am unable to say. The direct lateral cerebellar tract which conveys fibres passing to the cerebellum from the muscles of the trunk was also completely sclerosed. The cells in a great part of the posterior vesicular column were atrophied, and



there was no degeneration of the antero-lateral ascending tract; but on the question whether degeneration of the direct cerebellar tract is secondary to atrophy of these cells the case throws no light, for the crossed pyramidal tracts were also sclerosed, and Tooth has given reasons for believing that the cells of posterior vesicular column are connected with descending fine fibres running in these latter. Both Goll's column and direct cerebellar tract began about the usual level assigned to them. The degeneration of the crossed pyramidal tracts offers no features of any special interest; the absence of any lesion in the "mixed zone" of the antero-lateral columns may be connected with the absence of any affection of sensation.

The symptoms and pathological changes correspond generally with the disease known as ataxic paraplegia. Gowers states that it is rare for the direct lateral cerebellar tract to be affected. It has been considered doubtful whether ataxic paraplegia is to be classed among the diseases of the cord due to "system degenerations;" this case, at any rate, is a clear example of system degenerations, and the sclerosed areas are sharply demarcated from the healthy parts of the cord. If we may take the case as typical of others, ataxic paraplegia is due to a combined sclerosis of the crossed pyramidal tracts, affecting especially the fibres for the lower limbs, and of those fibres from the posterior roots which pass directly to the cerebellum from the lower limbs and trunk, the former in the posterior part of the column of Goll, the latter in the direct lateral cerebellar tract. The symptoms correspond to and are explained by this distribution of the lesion in the cord; unusual symptoms were the tremor of head and neck, the peculiarity of speech, the atrophy of optic discs, and the nystagmus during the last days of her life. Neither the optic atrophy nor the cord lesions can be considered as secondary to the extreme degree of anæmia which was present during the later stages of her illness, since they both existed for a long time before the anæmia supervened.

Elizabeth M., æt. forty-six, married. The following is a brief account of her case:—

She came of a healthy family, and had never had any bad illness; she had always been a temperate, hard-working woman. No history or evidence of syphilis could be obtained either with regard to the patient or her husband.

Her present illness began seven years ago with shooting pains in legs and lower part of trunk, which afterwards affected the arms and upper part of body. She was able to do her house-work until

two years ago. There was transient diplopia six years ago, which has occasionally returned and passed away again. The pains came with increasing severity and frequency. She suffered from well-marked gastric crises from time to time. For the last three years the legs have been very weak, so that she has had great difficulty in walking, and has been unable to walk unsupported for the past twelve months. There is a "woolly" feeling under the feet, and she has lost feeling in the legs and feet for the past two years. She also has suffered for some years from giddiness, worse in the dark; from girdle-pain for two years; from incontinence of urine, twelve months, and from obstinate constipation. Sight and hearing have been failing for three years.

On examination she is a tall, thin, anxious-looking woman. The thoracic and abdominal organs were normal.

There is great muscular weakness in the legs, and the muscles are small but not wasted. The gait is ataxic, she stumps her heels down on the ground. The knee-jerks are absent: plantar and lower abdominal reflexes also absent; the epigastric reflex present on the right side only; the triceps reflex present. Sensation over feet and lower half of legs is very defective, especially on the inner side of legs; all sensations are very much delayed; tactile sensation is more impaired than that to pain. There is also marked impairment of sensation from the above point up to the knees, it is deficient over the thighs but normal on the trunk and arms. No oculomotor paralysis; the left pupil is larger than the right; the right pupil acts to light and accommodation, the left to the latter only. There is pearly-grey atrophy of both optic discs; well-marked temporal hemianopia most evident in the mid-horizontal line, the line of demarcation extending close up to the fixation point. She can only read  $\frac{1}{2}$ -inch type at a distance of three inches; she is very deaf; smell and taste unaffected. Movements of arms slightly ataxic.

She was admitted into the hospital on account of the severity of the lightning pains, which were chiefly in the arms and shoulders, and on account of severe gastric crises occurring about once in every ten days.

She was now treated by suspension, beginning with a period of thirty seconds, and increasing by thirty seconds each time until a period of three minutes was reached. She was suspended fourteen times in five weeks. For three weeks she improved, was fairly free from pain and gained strength in walking, walking better for some hours after each suspension, and being able to walk a short distance without support. After this, however, im-

provement ceased, she constantly had severe lightning pains, and at the end of five weeks there came one of the violent attacks of gastric pain, sickness, and anorexia to which she was subject; this lasted five days and left her very weak. The first improvement in her condition was attributed to the more favourable conditions she was under in the hospital as compared with her home, and it was not thought advisable to resume suspension, from which, however, she had felt no ill effects, until she was stronger; as a matter of fact, suspension was not again carried out.

After this gastric crisis she was more comfortable for about a week, but had another attack of sickness, and now complained of pain in the lower part of the back and right hip. On examination the right hip and outer side of buttock were swollen, and a little later the swelling having increased and there being signs of pus, two incisions were made into the abscess and some unhealthy grumous-looking pus evacuated. The pus continued to be discharged, and a bed-sore formed over the sacral region and another smaller one over the right trochanter. Her temperature rose at times to  $100^{\circ}$  or  $101^{\circ}$ , she suffered from profound anorexia, from constant nausea and sickness, with from time to time lightning pains in the arms, and gradually sank and died about three weeks after the abscess was opened; her death being attributed to septicaemia and exhaustion from the discharge from the abscess.

The autopsy was made eighteen hours after death. The body was only fairly well nourished, the layer of subcutaneous fat being thin. The blood in the vessels remained quite fluid. The abscess described above was found to be situated in the right buttock and upper part of thigh. With the exception of the bed-sores above mentioned and the incisions into the abscess there were no scars or cicatrices on the skin. The heart was flabby and its cavities dilated; valves normal.

Some slight emphysema of anterior borders of lungs. Liver healthy. Spleen large, dark and soft. Kidneys: the capsule stripped badly, tearing the cortex in places; the cortex was somewhat narrowed, but otherwise the kidneys appeared normal on section. The walls of the bladder were thickened and there was chronic catarrh of the mucous membrane, which was slate-coloured; the ureters were dilated and contained turbid urine. The uterus contained five small intramural fibroids. Both brain and spinal cord were rather soft, attributed to the fact that an autopsy could not be obtained till eighteen hours after death. Membranes of both spinal cord and brain were normal and there were no adhesions. The brain appeared to be quite healthy in

all respects ; the cranial nerves looked healthy, with the exception of the optic nerves, which were small and wasted. The spinal cord was not cut across except for removing it, but reserved for hardening ; the section made on removal showed plainly degeneration in the posterior columns.

The spinal cord, medulla, pons, and basal ganglia were hardened in Müller's fluid, then for three days in spirit and stained by Weigert's process. The cervical region of the cord was unfortunately damaged in removing the cord, and was rendered useless for microscopical investigation. Sections of the motor area of the cortex cerebri and of the sciatic, obturator, and anterior crural nerves showed the normal appearances.

The following is an account of the changes in the cord :—

Sclerosis of posterior columns was more or less complete right through the coccygeal, sacral, lumbar, and dorsal regions of the cord.

*Mid-sacral region (fig. a).* There is complete degeneration of postero-lateral columns except for about one-third of their length immediately behind the commissure ; the sclerosis is most intense in Lissauer's tract, only about four or five undegenerated fibres entering the posterior horn from the posterior root ; there are few healthy fibrils in posterior horn, and in posterior corpus spongiosum ; but fibres in fair number pass into anterior corpus spongiosum from the inner side. At level of fifth lumbar the changes are much the same, but the undegenerated area in the anterior part of the columns is larger, and the plexus of fine nerve fibrils in anterior corpus spongiosum more abundant.

*Mid-Lumbar Region (Fig. b).*—Sclerosis of posterior root-zone (tract of Lissauer), and posterior roots almost complete, very few fibres escaping : in anterior third on right side and anterior  $\frac{2}{3}$  on left the postero-lateral columns show only partial sclerosis ; complete degeneration of postero - median column posteriorly, extending as a narrow band in the anterior half of the column on each side of the median fissure right up to the commissure. There now can be seen more numerous healthy fibres entering neck of posterior horn from inner side ; the fine plexus in anterior part of horn is well marked and some large longitudinally-running fibres are seen. Cells of postero-vesicular column few in number ; cells in anterior cornua deeply pigmented. In antero-lateral region is a thin band of partial degeneration running along the margin of the cord in front of the crossed pyramidal tracts. At level of first lumbar the changes much as foregoing, except that sclerosis is more advanced in front parts of postero-lateral and median

columns. At *twelfth dorsal* cells of postero-vesicular column are rather indistinct in outline, and protoplasm obscures nucleus, there are 12-14 on each side. At *tenth dorsal* (fig. c) they are more conspicuous, 12 on right side, 18 on left, some are deeply pigmented. There is diffuse sclerosis in antero-lateral region of cord, and the marginal zone of degeneration now extends further back towards posterior roots. In postero-median column degeneration extends to commissure, expanding in anterior third and narrowing again just as it reaches the commissure. The least degenerated part of posterior columns is a band bordering the inner half of the posterior horns. The previous description applies to the rest of the posterior columns. At *eighth dorsal* sclerosis of posterior columns most intense and extensive, the only area which is not destroyed is a narrow band behind the commissure and another just internal to the postero-vesicular column. The diffused sclerosis in the antero-lateral region is also most conspicuous here, chiefly as marginal zone extending from anterior roots in front nearly to posterior roots behind: there are besides some degenerated fibres scattered through the "mixed zone," and a few in the region of the crossed pyramidal tracts. About this point postero-vesicular column ends. Throughout its length the plexus of fine nerve fibrils normally present in it is atrophied, there is excess of connective tissue nuclei, the vessel walls are thickened with increased amount of connective tissue spreading from them; the fine longitudinally-running fibres which normally lie round the inner side of the column and those which should enter it are deficient in number. As to the cells they are in fair number throughout, some of them are deeply pigmented, in others the protoplasm is granular and the nucleus obscure, but others again look healthy and it is difficult to state positively that there is any marked change in them other than might be accounted for by the action of re-agents in preparing the sections.

At *level of fifth dorsal* (fig. a), postero-median and postero-lateral columns, with Lissauer's tract, show almost complete degeneration, with the exception of a narrow band of fibres lying between the two columns which has remained healthy in its entire length (a, fig. d). Above and at this level a fairly large number of healthy fibres now enter front half of posterior horn. The few fibres remaining undegenerated in the posterior root-zone are almost wholly of the large kind. The partially degenerated area in the antero-lateral tract is shown in the figure; there was besides some excess of connective tissue in position of crossed pyramidal tracts. At *second dorsal* (fig. e) condition much the same as fifth

dorsal, except that sclerosis is less intense in the postero-median, postero-internal and postero-external columns, especially in their anterior half. In sections taken from the fifth to the second dorsal inclusive, healthy fine fibres running longitudinally in the otherwise degenerated Goll's column are seen sparsely scattered through it. As above mentioned, sections of the cervical region were unfortunately not obtained. The vessels throughout the cord were dilated, and those in the grey matter and in the posterior columns were conspicuous from the thickening of their coats. Other regions of the cord not alluded to in the foregoing account appeared normal.

At junction of medulla with cord a sclerosed area borders the postero-median fissure and extends as a thin layer a short way along the posterior surface corresponding to the position of the *funiculus gracilis* (*y*, fig. *f*). An area of diffuse degeneration lies along the margin of the section in front of the tubercle of Rolando, but does not appear so plainly here as in a section taken at the level of the upper part of the decussation, where it is more distinct; its position is indicated at *x* in fig. *f*. At the lower part of the olivary body this tract seems to be concentrated as a narrow oblong sclerosed area lying at the margin of the medulla, with its anterior extremity just behind the olive and enclosed by external arcuate fibres, some of which course outside it. In sections above this at about the level of the glossopharyngeal nucleus (where the superficial auditory fibres first begin to appear), this tract could still be traced as a small oblong patch, much smaller than in preceding, lying at the margin more posteriorly, just anterior to the restiform body, nearly on a level with but outside the ascending root of the fifth; it was better marked on the right side, but this was probably the fault of the preparations (*x*, fig. *g*). Above this I could not detect it. The layer of posterior sclerosis at the commencement of the medulla extended upwards in the situation of the *funiculus gracilis*, and then occupied the superficial part of the grey matter next the median sulcus, as this opens out to form the fourth ventricle, and could be traced up for some way in this, gradually becoming thinner and disappearing (*y*, figs. *g* and *h*). Throughout the medulla the walls of the arteries in the grey matter were thickened and the vessels distended with blood (see fig. *i*). The vascular changes in the pneumogastric nucleus were very conspicuous, and could be distinguished when a power magnifying only three or four times was used. The region of the nucleus was densely occupied by distended capillaries and larger vessels, the finest capillaries being obvious by reason of their distension; the



lymphatic spaces around the vessels appeared full of blood-cells (possibly because they were filled by the distended vessel); in other places the spaces were empty but of abnormally large size. As to the cells of the nucleus, they had largely undergone atrophic changes, some being small and atrophied, others larger but with granular protoplasm and swollen rounded outline; in very few of the cells could the nucleus be distinguished, and nearly all were apolar. On carefully comparing sections from a healthy nucleus it was evident that the plexus of nerve fibres was deficient, that the fibres were all of fine size, whereas there should be many large fibres, and some of these fine fibres were varicose. The slender column showed distinct sclerosis, and the outer part of the pneumogastric nucleus showed similar changes. The figure represents the condition of the vessels and cells on the right side; atrophy of cells was equally or better marked on the left. The nuclei of the other cranial nerves were apparently normal. The vessels of the pons were dilated; the restiform bodies, superior olivary body, fillet and other parts of pons were healthy. The optic nerves were the seat of very dense old sclerosis.

To sum up, the parts in the cord chiefly affected were the posterior columns, and the ascending antero-lateral tract. Of the posterior columns, the tracts of Lissauer (internal and external posterior root-zone), were most completely destroyed, consequently the fibres that pass from the post. root into the horn were atrophied.

Speaking generally the anterior parts of the posterior columns suffered least. The few fibres that escape in the posterior root-zone were mostly of the large variety; the fine fibres seem to have undergone almost complete destruction. About the fifth dorsal an undegenerated tract comes into view lying between the posterolateral and postero-median columns, the fibres in it are of the small kind entirely, and run rather obliquely. As to its destination, at first sight it would seem to be commissural, but at about this level fine undegenerated fibres first appear in the column of Goll in considerable numbers and run longitudinally; it is possible that this may represent a tract containing fibres coming from the visceral or from the trunk muscles and running up to the medulla in Goll's column; further evidence is desirable on this point.

The degeneration in the antero-lateral tract was diffused, that is to say, consisted of degenerated fibres scattered amongst healthy ones, and was most advanced in the margin of the cord, in some situations forming a partial annular sclerosis. Being diffused in this way it is less distinct than the degeneration in the posterior columns, and must be secondary and later in time to this latter.

With regard to the course of this tract in the medulla, it would seem that the sclerosed fibres are gathered together into a well-defined bundle lying in the position indicated in the figure, and higher up probably pass backwards, at any rate in part, towards the restiform body. In the case of ataxic paraplegia where the direct cerebellar tract is completely degenerated and the ascending antero-lateral tract quite unaffected, the area of degeneration is not so well defined, but occupies as a diffused sclerosis the same situation in the lower part of the medulla just posterior and external to the olivary body, but could not be traced above this.

If morbid changes exist in the postero-vesicular column of cells, in this case of locomotor ataxy they are of slight and doubtful nature, there was at any rate no advanced atrophy of them. So far as negative evidence goes this would be against any connection of these cells with the fibres of the ascending antero-lateral tract. In the case of ataxic paraplegia they were atrophied in the lower parts of the column, and in the upper part had undergone extensive fatty degeneration; whether this atrophy is to be connected with degenerated descending fibres in the crossed pyramidal tracts, or with degenerated ascending fibres in the direct cerebellar tracts, cannot be determined in this instance on account of the presence of advanced sclerosis of both tracts.

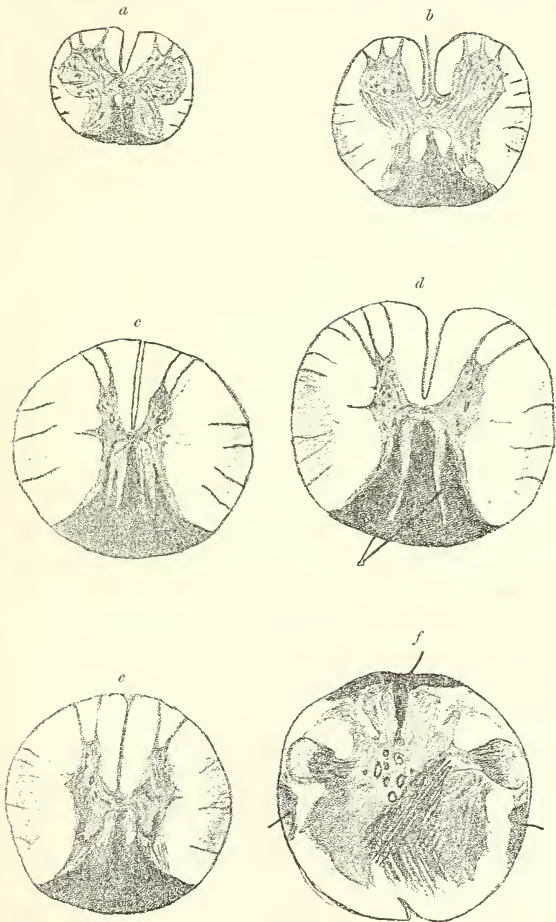
The lesion of the pneumogastric nucleus is interesting in connection with the severity and frequency of the gastric crises from which the patient suffered for so long a time.

It is difficult to understand in what way suspension can benefit a patient in whom the morbid process is so complete and extensive, and I am afraid that no light is thrown on the *modus operandi* of the treatment by this case.

In the figures the deepest shading is meant to indicate complete or almost complete sclerosis.

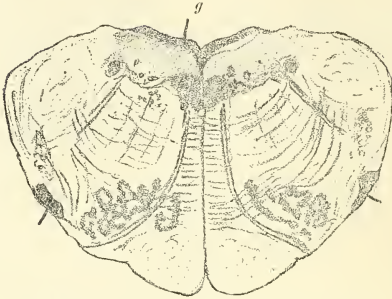


## DR. M. CLARKE'S CASE OF LOCOMOTOR ATAXY.



Sections at level of (*a*) Upper Sacral. (*b*) 2nd Lumbar. (*c*) 10th Dorsal. (*d*) 5th Dorsal. (*e*) 2nd Dorsal nerves, and (*f*) at Decussation.

## DR. M. CLARKE'S CASE OF LOCOMOTOR ATAXY.

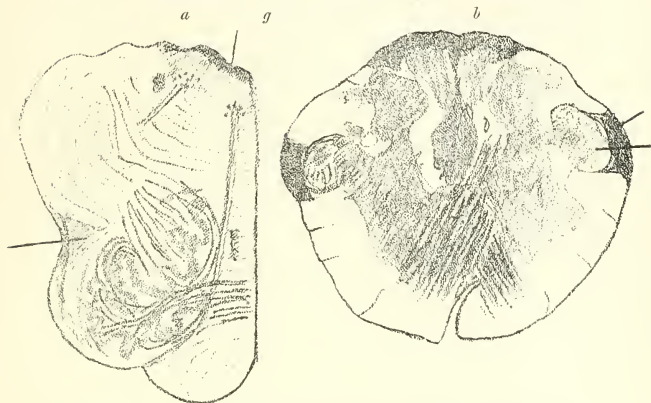


(g) Medulla Oblongata at level of Vagus nucleus.

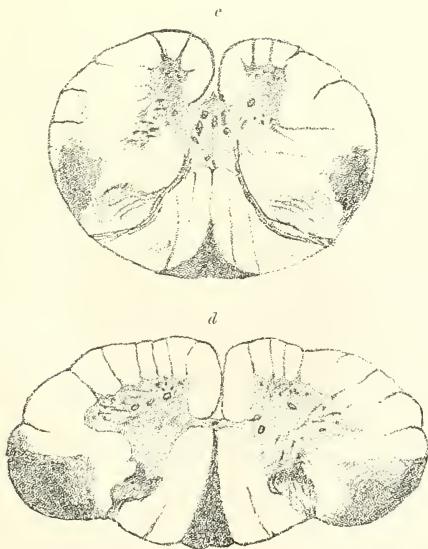


Locomotor Ataxy: Nucleus of Vagus on right side. (v) Vessels. (c) Pigmented and degenerated cells.

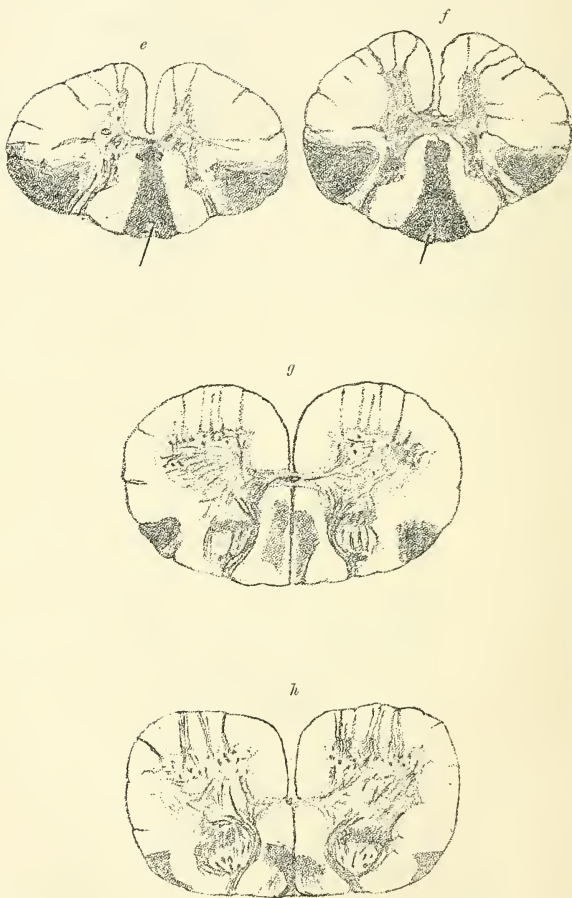
## DR. M. CLARKE'S CASE OF ATAXIC PARAPLEGIA.



Sections from (b) Decussation of pyramids. (d) Medulla Oblongata.



## DR. M. CLARKE'S CASE OF ATAXIC PARAPLEGIA.



Sections taken at level of (*c*) 1st Cervical. (*d*) 5th to 6th Cervical. (*e*) 3rd Dorsal. (*f*) 8th Dorsal. (*g*) 3rd Lumbar. (*h*) Upper part of Sacral region.

## ACUTE ASCENDING PARALYSIS (LANDRY'S DISEASE), FOLLOWED BY ATAXIC PARAPLEGIA.<sup>1</sup>

BY SANGER BROWN, M.D.

*Professor of Mental and Nervous Diseases in the Post-Graduate Medical School, Chicago, Ill.*

*Acute Ascending Paralysis* was first recognised and described by Landry in 1859. Since that time numerous supposed cases have been reported; though the points of similarity between some of the symptoms of the disease and acute ascending myelitis renders it somewhat doubtful under which heading some of the cases most properly belong.

The etiology is obscure, but remote influences correspond pretty nearly to those found in acute myelitis. The disease is more frequent in males than females, and chiefly occurs between twenty and forty. As an exciting cause, exposure to cold ranks first. Convalescence from small-pox, typhoid fever and diphtheria, have also been mentioned in this relation. Some authors regard syphilis as a frequent cause, but this opinion has not been generally received. No constant premonitory symptoms have been observed. General malaise, slight fever, pains in the head and back, formation, numbness in the feet and finger tips, have all occasionally preceded the onset.

The first definite symptom is usually weakness, beginning in the feet and extending steadily up the legs. It may begin somewhat earlier in one foot than in the other. The paralysis appears first in the muscles that move the feet, then those which move the leg, and later in those which move the thigh. It then extends to the muscles of the pelvis, loins, abdomen and thorax in the order named. The arms are next invaded, either from below upwards or from above downwards. The diaphragm and the neck muscles next suffer, difficulty of swallowing comes on and articulation becomes imperfect, or impossible. Unless the disease is arrested, the patient soon dies from asphyxia due to paralysis of the muscles of respiration. The sphincters may escape entirely

<sup>1</sup> Read before the Chicago Medical Society, March 3rd, 1890.

or suffer considerably. The sensory symptoms vary, from slight subjective numbness and tingling, to a considerable degree of anæsthesia. Sensibility to touch is frequently blunted, and that to pain and temperature changes may be delayed.

Pain is nearly always absent and never prominent. At first the cutaneous reflexes and myotatic irritability are lost; but in most cases which do not terminate fatally, it returns and may become excessive. Although the muscles soon become flaccid, they do not atrophy nor present altered electrical reactions, nor is there any tendency to bed sores, striking points of difference when the disease is contrasted with acute myelitis.

The cerebral functions are not involved. As a rule there is no elevation of temperature. Enlargement of the spleen has been observed in several cases. Though from two to four days is the period usually occupied by the disease, from its commencement till it ceases to progress, either from the supervention of death or otherwise, it may progress to a fatal termination in a few hours, or occupy so long a time as four weeks.

Though the order of invasion already described is most usual, considerable variation from it has been noticed. The arms have sometimes been involved before the legs, and bulbar symptoms have been said in some cases to take the precedence.

Very rarely there has been diplopia, paralysis of accommodation, dilatation of one pupil, and in one case double facial palsy. After having advanced rapidly to a certain point the disease may stop, the muscles last affected being the first to recover. Though the whole nervous system, including the sympathetic and the muscles, have been examined by the most competent pathologists, only negative results have been obtained.

The traces of inflammatory changes in the gray matter which a few writers claim to have found, have led them to pronounce the disease a form of acute atrophic paralysis. But the absence of fever, muscular wasting and changes in electric excitability are strongly opposed to this view.

The occasional enlargement of the spleen has led to the hypothesis of a toxic influence mainly acting upon that lower portion of the upper segment where its ramifications unite with the various cellular elements in the gray matter of the cord.

Now, so able an observer as Dr. Gowers makes disease of these filaments a working hypothesis by which the symptoms in this disorder and in ataxic paraplegia may be explained, and this case, so far as I am aware, the only one of the kind on record, would seem most peculiarly to confirm Dr. Gowers' hypothesis.

In reaching a diagnosis the rapidly ascending palsy, with relaxation of muscles and loss of reflex action, without fever, pain and loss of sensation, and if the patient survives without wasting of muscles or changes in electrical excitability are the main points to be borne in mind.

General ascending myelitis is the only disease which may present considerable similarity in the mode of onset and early symptoms. But in this latter affection there is always much disturbance of sensation, there is a strong tendency to bed sores and other evidence of trophic disorder; the muscles waste and will not respond to the faradic current, if the patient survives. A large majority of cases is fatal and the prognosis is always grave until the march of the symptoms is arrested. Relapses are infrequent.

As during the early stages the disease cannot be distinguished with certainty from acute myelitis, the treatment should be that most suitable for this latter disorder.

Rest, lateral decubitus, and counter irritation the whole length of the spine, are appropriate measures. Ergotin and salicylate of soda have each been employed with some apparent benefit.

*History.*—Following is a history of the case which has furnished the text for this paper:—A. B. æt. forty-two, married, native of Ohio, was brought up on a farm, and has been by turns, farmer, carpenter, and builder and merchant. Temperate, active habits and disposed to work long hours. Family history good. Mother still living, æt. seventy, and he thinks has cancer of the breast.

At the age of sixteen had a severe attack of pneumonia, and ten years ago a severe attack of typhoid fever, in the meantime, having had, while camping in the West, several severe attacks of malaria, during which he was delirious when the fever was at its height.

In the fall of 1883, forty days after exposure, had a small hard sore on the penis, which his physician pronounced syphilitic. No secondary symptoms were ever observed.

About six months prior to June, 1887, began to suffer with occipital headache, which never left him, and often manifested violent exacerbations, which increased in frequency and severity until towards the latter part of the period, when diplopia sometimes occurred at the climax of an exacerbation. The character of the pain, he says was steady, dull and heavy. He ate and slept fairly and was able to attend to business as usual.

Tried many physicians, and took various and numerous remedies, but without any relief.



On the morning of June 9th, 1887, travelled by train about twenty-three miles to consult a physician in regard to his headache, and while sitting in the physician's office about sun-down of that day, noticed a slight but distinct numbness of the toes of the left foot as if the parts were "asleep." About two hours later when he retired, the numbness had reached the ankle, but he noticed no weakness when walking. He awoke about 1 a.m. and thinking it might be time for an early train, which he wished to take in order to return home, he observed on going down stairs that the numbness had extended above the knee, and that the muscles of the leg were quite weak, so that he walked with difficulty, and at the same time he noticed that numbness had extended to the ankle on the other side.

When he rose the next morning at five, the numbness had extended to the hip on the left side and nearly to the knee on the right, and the weakness had increased considerably in both legs, so that it was only with great difficulty that he could walk to the railway station, a distance of about half a mile. When there he took a sandwich and cup of coffee for breakfast, but soon vomited, though he is certain that he had no fever at any time during the attack. He reached home at about 10 a.m. and found upon trying to walk a few yards to his house, that the weakness had increased very considerably, so that he needed assistance. Continued to grow weaker throughout the day, the numbness and weakness of the right leg having overtaken that of the left, progressed evenly above the hips, but more slowly than it had advanced up the legs. He slept some during the night, and next morning, by holding on to the fence along the walk for support, reached his place of business; but returned almost immediately, the paralysis having progressed very markedly in degree. About 10 a.m. the numbness having reached the umbilicus, severe nauseous vomiting and hiccoughing set in, and lasted pretty steadily for forty-eight hours. When the numbness had reached the umbilicus, it appeared in the finger tips of each hand, and extended nearly the whole length of the distal phalanges of the second and third fingers, and only at the extreme ends of the other fingers and thumbs. Here it ceased, and no weakness of the hands or upper extremities ensued, but the altered subjective sensation and blunting of tactile sensibility still remains as it does elsewhere.

The paralysis of the legs and lower trunk muscles appears to have been almost complete by the end of the first forty-eight to sixty hours, and it seemed safe to say that extension ceased at



the end of forty-eight hours from the commencement of the attack.

The patient is a man of active intelligence, and after the disease had fully developed, often tried to discover any possible power of motion in the lower extremities, and was only able to make very slight pressure against the bed clothes with the outer side of each foot.

The sphincters were distinctly affected but not greatly. Haste had to be made when he felt the desire to urinate or defecate—if the passage was loose—in order to prevent an accident. Constipation resulted probably from lack of exercise and paralysis of abdominal muscles.

The mind was clear so that the patient kept the run of his business as perfectly as ever, but he felt a dislike for his wife and children which he had never felt before. Did not want them about him, and wanted the constant presence of his mother. This may have been nothing more than an excessive degree of the peevishness often observed in invalids; the feeling subsided as improvement began.

Sexual power was lost, though desire was not much altered. First began to regain some power of movement in bed in about six weeks from the onset, and by the end of two months could stand by supporting himself by holding on to the backs of two chairs.

I am unable to get any information regarding the state of the reflexes while the patient was in bed, but the cutaneous reflex and ankle clonus were well marked when he began to walk, as well as spastic symptoms.

While sitting in a chair with the ball of the foot on the ground and the heel unsupported, marked clonus would be produced. If he would step with his bare foot on a small piece of coal, would be thrown down by the violence of the contraction thus produced, and his legs would become rigid when he suddenly straightened them out in bed.

Incoordination was first noticed a few months after he began to walk, though he thinks it may have existed earlier. Inability to walk in the dark first attracted the patient's attention.

Patient thinks that on the whole there has been a sort of interrupted improvement, extending from a period about six weeks after the onset down to the present time, though slower within the last year than before.

The subjective influence of custom, and the increased facility gained by practising locomotion under difficulties must all be

taken into account in considering the patient's estimate. He has, however, been for nearly six months past under the observation of my assistant, Dr. Slater, at the Post-Graduate Medical School, who has watched and studied the case with great care, and he reports distinct improvement within that period.

*Present Condition.*—Patient is well nourished, and, with the exception of a somewhat capricious appetite, his general health is good. Probably owing to some weakness of the abdominal muscles, and some blunting of sensibility of the rectum, he has to stimulate this latter organ by the insertion of his finger, in order to effect a movement of the bowels. The sphincters are still somewhat weak, as he says it would be difficult, or even impossible, for him to retain a loose passage after taking physic, and he has to make haste when he feels the desire to urinate.

The gait is typical of ataxic paraplegia; the feet are kept far apart; the legs are rather dragged after the body, and the toes scrape the ground. There is considerable jerkiness in any movement of the leg. Patient cannot stand with the eyes closed.

Myotatic irritability, as evidenced by the knee-jerk and ankle clonus, is considerably exaggerated, and so is the cutaneous reflex from the soles, though it is not developed by a slight touch. A slight prick of a pin produces a sharp contraction. Cremasteric, gluteal, and abdominal reflexes could not be obtained.

There is no muscular atrophy in any situation, no tendency to trophic changes, and electrical excitability to both currents is normal in quality, though possibly reduced somewhat in quantity. The patient thinks the subjective feeling of numbness—exactly as if the parts were asleep—is the same as at the time of the onset.

The sensibility to touch and temperature changes is distinctly blunted. When a test tube filled with hot water was firmly applied to any part, he promptly responded correctly, but when a tube of exactly the same size and shape filled with cold water was applied, there were many parts below the knee upon both legs where he could not be certain if it was hot or cold, and in an oval space, about three inches in the longest vertical diameter, on the inside of the left calf, he frequently pronounced a cold body hot. The prick of a pin produces pain promptly, and he thinks natural in quality.

Tactile sensibility, and sensibility to temperature changes, improves gradually from the knees to the upward limit in the vicinity of the umbilicus, which comprises a zone of doubtful territory about two inches wide. That is when the finger is passed slowly either upward or downward, and the patient is requested, with

closed eyes, to indicate when the limit is reached, he will say, "right about there," when the finger is arrested anywhere in a zone as above described. Though, while the patient has been under my observation, the left pupil has generally been a trifle larger than the right, I have been unable to discover any conspicuous defect in vision, movement, the fundus, or ocular reflexes, that would tend to throw any light on the case.<sup>1</sup>

I should have stated that throughout the period of headache preceding the onset of paralysis, the patient had ulceration of the scrotum, which itched intolerably, and which he habitually scratched, when the paralysis supervened, his attention was no longer directed to the scrotal itching, and the ulcer healed, leaving a cicatrix which I would not be able to pronounce positively specific.

An acute process commencing at the lower extremity of the lumbar enlargement, and rapidly progressing to its upper extremity—or to the 11th dorsal pair slightly above the upper extremity—and there ceasing, and then commencing between the 7th and 8th cervical pair at the bottommost part of the functionally important region of the cervical enlargement, and manifesting the same progressive tendency that was shown in the lower segment, would seem to lend peculiarly strong support to the etiologiocal hypothesis put forward by Gowers, that this disease may be due to a toxic or other influence acting upon the fine filamentous terminations of the upper segment in the grey matter of the cord.

That the process stopped near the level of the 11th pair to continue near the 8th. If possibly it did not begin in the latter before it had entirely ceased to progress in the former situation—would indicate a special susceptibility of the parts in proportion to their distance from their centre—the brain—their functional complexity and importance, and the distance from them and from their centre, of the parts to which the special modes of energy they severally convey is ultimately conducted.

That is, it would be easy to understand how a comparatively coarse mechanism might suffice to distribute the necessary energy to the abdominal muscles, while an exceedingly fine and complex mechanism might be required to supply the hand, finer, indeed, than that for the foot, but the increased distance of these latter filaments from their centre might give them the priority in point of vulnerability.

<sup>1</sup> My colleague, W. Frank in Coleman, M.R.C.S., after careful examination, finds varying insufficiency of the internal recti.

I shall refrain from attempting to account for the nausea vomiting, and hiccoughing, as well as the antecedent headache.

The mode of onset of the ataxic paraplegia in this case, together with the fact that the course has been regressive instead of progressive, removes it from the category of ataxic paraplegia proper, yet the present symptoms are highly typical of that disease.

## ON PARALYSIS OF THE THIRD NERVE AS A COMPLICATION OF GRAVES' DISEASE.

BY JAMES FINLAYSON, M.D.

*Physician to the Glasgow Western Infirmary, and to the Royal Hospital for  
Sick Children, Glasgow.*

A MARRIED woman, 36 or 37 years old, was admitted twice to my ward in the Glasgow Western Infirmary (May, 1888 and November, 1889), with what I could only regard as Graves' disease, even although, as sometimes happens in this affection, exophthalmos was absent,<sup>1</sup> and no history of its presence could be ascertained. When about 33 years old, she became very nervous and easily disturbed, with palpitation on excitement or exertion. Two and a-half years later, April, 1888, she was suddenly seized with a sensation of blocking of the windpipe, and at the same time a swelling appeared there, especially noticeable in the right side of the neck. On admission to the ward, next month, this was found to be a goitre. The swelling was characterised by throbbing and pulsation: its degree of fulness varied much from time to time during her residence: it was quite plain, but not obtrusive. She had also a quick pulse, and there was pulsation in the epigastrium, with a soft blowing systolic murmur over the heart. There had been repeatedly attacks of sickness and vomiting, with pains in the stomach. Some of these attacks were of a severe character during her second residence in the ward. Her menstruation had ceased in January, 1888; she had had her last child some two years before that. She had latterly been exposed to a good deal of hard work and privation. Her family history seemed to have little bearing on her case, unless for the death of her father, at 40 years of age, from heart disease. Her case was complicated by the occurrence of otitis media, first in the left ear, nine months after her last child was born, and a similar very painful affection, in the right ear, appeared during her second residence in the ward: so far as appeared, these attacks were of an accidental character, and had no connection with her other disorders: they were asso-

<sup>1</sup> "In some few cases the exophthalmos is absent."—Dr. Ross; *Treatise on the Diseases of the Nervous System*. Second ed., vol. i., p. 712. London, 1883.

ciated with ceruminous accumulations, and perforations of the membrana tympani.

Coming now to the eye symptoms, it may be said that no indication of present or past exophthalmos could be traced. On her first admission, in May, 1888, there was a very notable, although not complete, droop of the right eyelid. She reported that she had had severe headache, one night in May, 1887, about twelve months before admission, in the right temporal region, and next morning she was unable to open the right eye. This droop persisted more or less, although during her second residence it was not quite so noticeable. On further examination, the right pupil was found dilated, and there was no contraction with light or on attempting to accommodate. There was an extremely marked divergent paralytic squint of the right eye, and diplopia could be made out in certain directions. There was marked immobility of the right eye in all directions except outwards; all the muscles seemed paralysed except the external rectus, and perhaps the superior oblique. The visual acuteness in both eyes was diminished, especially in the right. On ophthalmoscopic examination, nothing but a certain pallor of the discs was found by Dr. T. Reid. There was no paralysis of the face, and her sense of smell and taste seemed good. There was thus an *isolated paralysis of the third nerve* on the right side, involving the movements of the eyeball and of the iris, and partially involving the elevation of the upper eyelid. She improved considerably during both residences in the hospital, but the treatment need not be detailed here.

During her first residence the peculiar paralytic symptoms in the eye seemed to me quite different from any complication of Graves' disease of which I had ever known; and I then made some search into the literature of the subject. I failed to find anything bearing on the matter except a paper by Dr. Bristowe, entitled "Cases of Ophthalmoplegia complicated with various other affections of the Nervous System," published in *BRAIN*, vol. viii., 1886, p. 313, and afterwards reprinted by him in his "*Clinical Lectures and Essays on Diseases of the Nervous System*," Lond., 1888, p. 69. It appeared that the illness of the same patient had been reported by Dr. Francis Warner; "Ophthalmoplegia externa, complicating a case of Graves' Disease;" "*Medico-Chirurgical Transactions*, vol. lxvi., Lond., 1883, p. 107. In my case, however, the paralysis was of the third nerve and not an ophthalmoplegia, and it differed in other important respects also from Dr. Bristowe's.

On the second admission of my patient, I discovered an im-

portant article on this subject, by M. Gilbert Ballet, in the *Revue de Médecine*, for 1888, pp. 337 and 513. Its title is “L’Ophthalmoplégie externe et les paralysies des nerfs moteurs bulbaires dans leur rapport avec le goitre exophtalmique et l’hystérie. Contribution à la physiologie pathologique de la maladie de Basedow.”

In this paper a great many cases are brought together (including Dr. Bristowe’s), and the subject is discussed in a very interesting manner from the point of view of a paralysis of the bulbar nerves in these affections. The author asks (p. 535) if, apart from the generalised form of paralysis termed ophthalmoplegia, a partial paralysis involving the ocular nerves (3rd, 4th, or 6th pair), has been observed in exophtalmic goitre? He can only quote one definite case of this kind, although in some cases impaired movements of the eyeball had been observed, apparently due to the mechanical effects of the exophtalmos: this case was by M. Féréol, and in it the 4th nerve was involved.

The case here narrated, seems therefore to have a certain value in supplying very definite proof of the implication of the 3rd nerve in this remarkable disease.

## NOTES OF A CASE OF PROGRESSIVE NUCLEAR OPHTHALMOPLEGIA.

BY W. M. BEAUMONT.

*Surgeon to the Bath Eye Infirmary.*

NURSE P., aged 41, was sent to me in Nov., 1889, suffering from ophthalmoplegia. She gave the following family history:—Her father died ten years previously of chronic bronchitis, aged 73; her mother died seventeen years ago of apoplexy; one brother died of consumption, one of inflammation of the lungs, and several others died young. One brother living suffers from disease of the heart. One sister died of scarlatina, in infancy; a second sister, living, is a martyr to rheumatism (probably rheumatoid arthritis), and a third sister occasionally suffers from rheumatism. There is no definite history of gout in the family.

*Past History.*—She remembers when she was very young, in fact it is one of her earliest recollections, being told to look up at the ceiling without moving her head, and being laughed at for failing to do so. About the age of ten left ptosis was noticed, and continued for seven years, being usually worse in the spring than at other seasons; it was then cured by operation at the Bath Eye Infirmary, and has not since recurred. Nothing was noticed with regard to the right eyelid at this time, but two years later, when she was 19 years of age, she began training as a nurse. It was then observed by her that when she was very tired the right upper eyelid drooped a little; later on it became more noticeable, but always disappeared when she rested. At the same time she occasionally suffered from diplopia, and the light dazzled and confused her. To obviate the annoyance of these symptoms, she resorted to tinted glasses when out of doors. The glare from the sea, or from snow, was especially trying. After six years hospital nursing she went to live in the country for six years (1873-1879), and was not troubled by her eyes during that time. In 1880 she began private nursing, and almost immediately the ptosis of the right returned, and has remained permanent since. It is usually intensified by night-work, and by anxiety connected with critical cases.



Apart from her eyes she has never had much to complain of in her health. There is no history of acquired or hereditary syphilis. She had an attack of scarlatina at the age of 17, and since then occasional attacks of "swimming in the head," also headaches at times, which are usually more severe on the right side of the head. She has never been a good sleeper, lying awake for hours together without any assignable reason. No history of "fits." Never suffered from œdema of face or eyelids, and there has never been any divergence of eyes nor proptosis.

*Present Condition.*—The pupils measure 5 mm. in diameter each; they act moderately and equally to accommodation. The right responds very slightly to light, the left rather more so, and both fully dilate with atropine. The fields of vision are contracted downwards, but normal in other meridians. To look in any direction outside her field of vision it is absolutely necessary for her to move her head, the eyes being completely stationary, and apparently quite incapable of movement in any direction. Recti and obliqui are alike paralyzed. There is well-marked ptosis of the right lid, giving that side of the face the sleepy expression so usual in such cases. The refraction of the left eye is emmetropic, in the right there is a moderate degree of compound myopic astigmatism. Vision, R.—1·5 sp.  $\bigcirc$ —1·5 cy. =  $\frac{6}{6}$ . L.  $\frac{6}{6}$ .

There is no deafness, no loss of sensation anywhere, and the knee-jerks are vigorous. No ankle-clonus. Sense of taste and smell normal. She has been married for a few months, but there has been no alteration in her symptoms since.

*Remarks.*—The principal point of interest in this case seems to be the prolonged duration of the disease, certainly over more than thirty, and probably more than forty years. It would appear to be congenital, and if syphilitic in origin, it is so by inheritance. Apart from the occasional headaches there appears to be nothing that would point definitely to disease of the cavernous sinus, to periostitis or to a peripheral neuritis, for there are no complications; the general health is good and the fifth nerve is not involved. It has been slowly progressive with long intervals of quiescence, indicating probably a nuclear origin just posterior to the centres for accommodation and pupil-contraction.

There has been no opportunity of trying the effect of drugs on her condition, as she does not consider herself ill enough to submit to treatment.

## A CASE OF SPASTIC SPINAL PARALYSIS— TREATMENT—RECOVERY.

BY E. DUTOIT, M.D. (BERN).

ALFRED KLÖTZLE, æt. 21 years, was a farm-servant, and in all respects healthy until the 10th November, 1883, when he fell from a height of from 12 to 15 feet upon his heels. Noticed nothing peculiar about himself for the next few days, during which time he was a good deal exposed, working in his shirt-sleeves in the fields, the weather being cold and damp. On November 18th, he for the first time experienced pains in the hands and feet. These continued, and weakness of the legs developed to such an extent that he became soon unable to walk, and was admitted to hospital on December 1st, 1883. On admission, slight wasting of the calf-muscles was noticed, more marked on left side than on right side; they were also somewhat contracted, so that some effort was necessary in order to move the feet, especially the left foot. The patient found difficulty in walking, being unable to bring his heels to the ground, and progressing upon the balls of the toes. Sensibility of skin everywhere normal. Faradic and galvanic reactions—both of nerves and muscles—normal. Knee-jerks increased, especially that of left side. On elevating the foot, tremor of the muscles of the upper and under aspects of the thigh become manifest on either side. Temperature and pulse normal: appetite good: bowels regular: urine normal. Patient was prescribed *argent. nitrat.* 0·01 grm. three times daily. Aggravation of the above-mentioned symptoms was noticeable up to the middle of December, 1883; the tendon-reflexes became more brisk; the contraction of the calf-muscles advanced to such a degree that the patient could barely walk, even on the balls of the toes. A more powerful and prolonged effort was necessary ere the foot could be moved, and it was accompanied by much tremor. On December 20th, treatment with the galvanic current was commenced, the anode to the nape of the neck, cathode to the lumbar region: strength of current, 10 to (at most) 15 milliampères; duration of the sitting, 3 to 5 minutes, once a day. On January 3rd, 1884, as the pains in the calves steadily increased, the feet were fixed in a position at

right angles to the legs by means of plaster of Paris bandages reaching to the knees. Patient was at once relieved. The mode of application of electricity was altered at this time; the sitting was prolonged to a period of from 6 to 8 minutes, and the direction of the current was suddenly reversed in the course of the application. At the end of March the bandages were removed. The atrophy of the calf-muscles had increased. At the end of 5 or 6 hours the contraction of the muscles and the pains in the calves reappeared. Walking now required greater effort than ever. Electrical reactions normal. Fresh plaster bandages were put on at the beginning of April. At the close of May these were removed, and it was found that the atrophy of the calf-muscles had still further progressed. Walking now almost impossible. Tendon reflexes much exaggerated. Commencing contraction of the flexors of the knee. Slight diminution in faradic reaction: otherwise no electrical change. Severe effort necessary to raise the feet, and much tremor produced thereby. The bandages were renewed, reaching to a point above the knee. Galvanisation continued to end of June. During July, August, and September, it was discontinued (being recommenced on 1st October). The silver nitrate was omitted on June 1st; bandages also discontinued for from two to three months. At the end of this period the atrophy, tendon-reflexes, tremor, contractions and electrical reactions were unaltered. New bandages were therefore put on and galvanic treatment recommenced, the strength of current being sometimes increased to 18 milliamperes. On removal of the bandages at the beginning of January, 1885, a slight improvement was for the first time noted. The tendon-reflexes, the tremor on raising the feet, were diminished in degree, faradic irritability had increased; the calf-muscles were of greater bulk. No fresh application of bandages. Patient still walked on the balls of the toes.

Early in March he began to complain of the severity of the electrical treatment; the strength of current was therefore gradually lowered to 12 milliamperes. In April the knee-jerks were found to be normal, and no further tremor on movement was observed. There remained only a certain tenseness or stiffness in the region of the calves. Progressive improvement took place in the general condition, and in that of the muscles. Patient was able to use both feet properly. Electrical treatment discontinued at end of May, and patient was discharged on June 6th, 1885. He returned to his former occupation.

On Nov. 6th, 1885, K. applied for re-admission, on account of

the reappearance of all his former symptoms—in slight measure. Some rigidity of the calf-muscles and slight increase in tendon-reflexes were noticed: in walking the heels did not quite touch the ground. Slight tremor on raising a foot; he also complained of smart pains in the calves. Electrical reactions normal. Patient had been working in the open again in his shirt-sleeves during cold weather; had been drenched with rain, and had neglected to change his clothing. Electrical treatment was at once renewed, the strength of current being from 12 to 16 milliamperes; duration of sitting, from 6 to 8 minutes. The current was reversed during the sitting. By the end of a month all the symptoms had disappeared, excepting the stiffness or rigidity of the calves. Galvanisation was continued three times weekly until the end of April. Patient was now enabled to stand the whole day without fatigue, noticing merely that his left leg became somewhat quickly tired. He obtained a situation in the hospital, his main duty being to go errands, so that he was about for three or four hours daily. He continues in this post at present time, and gives no evidence of return of his malady.—[*Translated from the original German MS. by E. W. GOODALL, M.D.*]

## Reviews and Notices of Books.

*The Functions of the Central Nervous System and their Phylogenesis.* Second Section—Fishes; with twenty-seven woodcuts and one lithograph. By Prof. Dr. T. STEINER (Heidelberg). (Braunschweig, Vieweg and Son, 1888).

IN the first section of this work, which was devoted to the brain of the frog, the author laid down a general plan of the action of the central nervous system in that animal, and he now passes on to similar researches in the class of fishes. While he keeps closely to the established facts of comparative anatomy, and everywhere endeavours to utilise them, not only functions may be studied, but also their phylogenetic development. In the following *resumé* we shall have the opportunity of seeing what advantage such a method possesses.

The book is divided into ten chapters, in which all the great classes of fishes are considered, in as far as they concern the subject in hand. In the FIRST CHAPTER we find a research upon the function of the fins and upon equilibration, with the object of deciding the mechanism of movement and the equilibration of fishes. The fins are glued to the body with warm gelatine in such a manner as to prevent their action. Observations upon finless fishes, as well as upon those fishes whose fins have been rendered incapable of function in the manner described, prove that the fins do not serve fishes for locomotion, but that it is accomplished by the to-and-fro pendulum oscillations of the tail (with the exception of those fishes which move by a serpentine action of the body).

Fins have not the task of maintaining equilibration of a fish when engaged in rapid movement, nor of the same fish when it remains on the bottom. But the unpaired fins are indispensable for the free suspension of the fish at any height in the water, and one never sees fishes which have no paired fins, as *e.g.* lampreys, freely suspended in water. Further, these fins serve as a rudder

for the stopping of quick movements, and finally for backward movements. The equilibration of the fish does not depend upon the fins, but is a function of the muscular sense, which by reflex channels brings about the necessary correction in equilibration for every displacement which occurs. This theory has been more precisely developed in the first section (Frogs) for all vertebrates ; in its wider sense it applies also to invertebrates.

The SECOND CHAPTER contains an account of the osseous fishes. Previous experiments upon the brain of the fish all suffer from the same fault, namely, that the skull once opened could not be closed again, and the free surface of the brain was thus exposed to the injurious influence of the water washing around it. In order to be able to close the skull cavity again the author makes use of the following method:—The skull cap is carefully raised to some extent but left in connection with the body of the fish posteriorly as a bony flap. After the operation on the brain is finished the bony flap is again replaced, fastened in front with a thread, and warm gelatine poured over the wound, and this gelatine cap is painted over with concentrated solution of tannin. In this way the cranial cavity is completely closed, and when the gelatine cap falls off in about two days, the bony flap is firmly fixed in position. The whole process is carried on with artificial respiration, and the bony fish used is *Squaliuscephalus* (v. Siebol), a common cyprinoid in the waters of middle Europe. After the fore-brain (cerebrum) has been removed with these precautions, it will be seen, that the movements are perfectly normal, and that the fish sees, since it avoids obstacles, facts already known. But as is now evident, the movements are not altogether automatic and constrained, but appear entirely voluntary, since the fish now poises itself at any height in the water, now rests on the ground. *It is, however, perfectly new and unexpected to find that this fish one day after the operation catches an earthworm thrown to it and swallows it in the usual way.* But if a thread of similar dimensions to the earthworm be thrown, it will not be taken by the fish. Later on such fishes take small pieces of bread or "*blatta orientalis*" from the surface of the water. If different coloured wafers are thrown to them, the red ones are always preferred ; other colours make no especial impression. Finally, it is necessary for the success of this experiment to have a tank with *flowing* water ; it was not successful in a tank of still water. Osseous fishes operated upon in the above manner may live an unlimited time, like normal fishes. Vulpian confirmed these experiments a few months later upon carp.

These results can only be understood by supposing that the great brain has been an organ once endowed with functions, but that in the course of development it has lost its functions; anatomical research upon this subject demonstrates also that the roof of the great brain of the osseous fishes, the mantel, is absent (Rabl-Rückerd). This defect is, however, not the cause of the loss of function, but as will be shown later on, only a sign thereof.

As already known, removal of the roof of the mid-brain makes the fish blind. The author has far surpassed his predecessors in the removal of the cerebellum, since he was able to scoop out not only the free portion of the cerebellum but also the anterior part of the same situated in the mid-brain cavity (*valvula cerebelli*), without any disturbances in movement being observed—at least as far as movement in a straight line and resting upon the bottom are concerned. If the base of the mid-brain be removed, the fish at once falls on its back, and only mechanical excitation can cause movements; placing the fish in the water will no longer incite it to movements. Locomotion, moreover, ceases definitely if the most anterior portion of the medulla behind the ganglion impar is divided. The lobus impar is a structure peculiar to the osseous fishes which rises out of the medulla, and which, as experiment shows, contains the respiratory centre. In these fishes, as in the frog, there is a point situated in the anterior portion of the medulla which serves the animal the purpose of locomotion and which we have termed the common or cerebral centre of movement. And just as in the frog we have here to look to the base of the mid-brain for the central station of stimuli coming from the skin, muscles and joints, and conveyed to the medullary centre in order to excite movements. Consequently in fishes the whole of this area of medullary and mid-brain centres which have, however, no definite anatomical boundary, will in future be designated by us as the common centre of movement. This centre is not anatomical but of a reflex nature, and may also be excited by impulses conveyed by the higher sensory nerves.

The THIRD CHAPTER is devoted to the amphioxus, which can be cut into two or three pieces and each portion will balance itself and execute co-ordinate movements, with the head end foremost. It is therefore probable that the amphioxus possesses no common centre of movement, but that the amphioxus is composed of a number of perfect similar locomotor metameres.

The FOURTH CHAPTER deals with sharks, which have for the first time been investigated by experiment. The experiments



were carried out at the Biological Station of Naples in the spring 1886-1887, on several species of Scyllium.

If the fore-brain is removed in these fishes just as in the osseous fishes, no disturbance of movement is observed. But with the spontaneous seizing of food there is a difference, for they never seek the sprats thrown into the tank which they are especially fond of, consequently sooner or later they die of starvation. The same result is obtained however in sharks if, instead of removing the fore-brain, its connection with the central organ of smell—the lobus olfactorius—be cut through on *both sides*, whereas section on *one side* does not destroy the capacity of the fish to seize food.

Sharks behave differently to osseous fishes in this respect, and again differently to amphibians, *e.g.*, frogs. We shall refer more particularly to this in the generalisation. The removal of the thalamencephalon (Zwischenhirn) produces no disturbance of movement, but it appears that when the fish is excited to movement it comes to rest sooner, that is to say, the inclination to move is somewhat lost.

Removal of the mid-brain of the shark produces corresponding results to that in the osseous fishes, only the tendency to movement is more marked, and therefore it is with more certainty demonstrated that simple contact with the surrounding water produces no excitation which will lead to movement.

The removal of the cerebellum in this case causes no disturbance of movement or balance. A section through the medulla, above the origin of the vagus, causes the shark to lose power of movement. If however, the spinal cord is divided below the exit of the vagus or still lower, even if the animal is simply decapitated, the hind portion apparently performs perfectly normal and co-ordinated movements, which may continue for upwards of an hour or more, and under certain circumstances even for a day.

Close to the common centre of movement, situated in the mid-brain, we find also a spinal centre of locomotion, which can be proved to be subordinate to the former, which directs all movements of the animal. The advantage which is possessed by an animal having thus, by a process of evolution, acquired such a common centre of movement lies in the fact that it is able to move easily in any plane, whereas animals without that centre, in which single metameres are capable of co-ordinate movements, are only able to move in *one plane*.

Experiments upon the spinal cord of rays (*torpedo oculata*), sturgeons and lampreys, are undoubtedly of fundamental impor-



tance when taken in consideration with the results of experiments upon the spinal cord of the shark. The spinal cord of rays and sturgeons acts just the same as that of the shark, but the spinal cord of the lamprey after decapitation of the animal can evoke no locomotion, when the fish is put into a bath of 1—3 per cent. picro-sulphuric acid.

The functional capacity of the spinal cord of the eel is interesting; after decapitation the hindermost portion possesses similar motile powers, whereby under certain circumstances actual movements from one plane to another may be observed.

The NINTH CHAPTER treats of forced movements, which follow exactly the same rules as in the frog. Unilateral injury of the base of the mid-brain causes a circus movement towards the uninjured side. Unilateral section of the medulla produces rolling movements towards the injured side. The pleuronectidæ among the osseous fishes exhibit an especially interesting condition by the fact that the circus movement takes place in a vertical plane, which might be predicted on account of the manner in which these fishes have come to swim on one side instead of on the belly. Of essential importance is the fact that after unilateral injury of the spinal cord the shark, although capable of locomotion, is never able to perform forced movements. In spite of this, it is worth an experiment to force the spinal cord to produce circus movements. If a shark is set into circus movements by removal of one side of the base of the mid-brain and allowed to remain thus for ten hours at least, then decapitated, the trunk will still make circus movements and in the same direction as before decapitation. Taken with many other interesting results, this experiment proves in the clearest manner that the common centre of movement in the brain governs the centres of movement of the spinal cord. If unilateral injuries of the brain are compared with bilateral, it will be observed that by unilateral injury only those parts of the central nervous system which stand in immediate relation to the centre of locomotion yield forced movements. The forced movements are a function of the common centre of movement whereby it is to be concluded that where forced movements are found a common centre of locomotion is present, and *vice versa*. At the same time it may be observed that forced movements form the simplest and most certain evidence of the common centre of locomotion being present.

The TENTH AND LAST CHAPTER contains the general conclusions yielded by the facts communicated. In connection with

the morphology a definition is given of the separate parts of the fishes' brain, which had formerly offered many difficulties of explanation. However, on this part of the subject the original must be referred to, because it cannot be clearly explained without the diagrams.

A detailed statement upon the plan of the *great brain* in fishes now follows. It has been shown above that the removal of the fore brain, and the separation of the central olfactory organ yielded the same result in the shark. *It follows from this that the great brain of the shark consists of nothing else but an olfactory centre.* In fact, the sense of smell takes that place in the life of the shark which belongs to the great brain in other animals, and it is only the sense of smell which guides the shark even by day to its prey and enables it to obtain its food. Since the great brain of every vertebrate is homologous, it further follows, *that the great brain of the vertebrate has developed phylogenetically out of the olfactory organs.* But if a higher sense centre, such as that of smell, can take on the functions of the great brain, we must ascribe this power also to other centres, especially to the visual centre. We actually see this to be the case in osseous fishes, where the great brain has degenerated and the visual centre has taken its functions.

In conclusion, the author has undertaken the task of determining the genealogy of fishes upon physiological data, which he endeavours to solve in the following manner. Morphology points to the selachia (Elasmobranchii) among the now living fishes as the original parent fish. We have, therefore, to consider the functions of this animal as the original functions from which those of all other fishes and vertebrates are derived. The central nervous system of the shark consists of brain and spinal cord, which parts are however only treated from an anatomical point of view, in order to lead up to the physiological definition.

There is no absolute definition for the brain, but by comparing all classes of animals, and by the help of that general proposition concerning forced movements the following definition may be formulated:—*The brain is defined as being the common centre of movement in connection with the functions of at least one of the higher sense organs.* Where both these conditions are not co-existent, a brain cannot be considered to exist.

Accepting this definition, can the relationship of the brains of vertebrates be determined by comparison? No, for none of the original properties of the brain have been lost in later developed vertebrates. We only see here a further development of

some function already present from which no inference of relationship can be drawn.

We turn therefore to the spinal cord, whose single function is one of locomotion, which function consists in the co-ordinate action of all the metameres, and which we have to consider as an original function. If we herewith compare the spinal cord of other vertebrates, we find that this function is sometimes lost, and in proportion thereto, the greater or less departure of the animal in question from the parent stock of vertebrates may be inferred.

We may now form the following idea concerning the phylogenetic development of the vertebrata. The stock of the vertebrates commences with an acrania, a vertebrate consisting of similar metameres, for which the well-known amphioxus lanceolatus serves as a type. This primitive condition may be physiologically expressed by the fact that all the metameres possess the same function of locomotion, by which the animal moves itself in one and the same plane in the easiest manner. In a certain phylogenetic period the metameres commence to develop their power of locomotion in front, by which the anterior metamere is so much strengthened that it takes the lead of the others.

The objective evidence of this departure is a migration of the function towards the anterior end. The proof that the most anterior metamere has taken the lead lies in the fact that only unilateral injury of the central nervous system of this metamere is able to transform the movement which, previous to the injury, was in a straight line into that of a curve. The centre of one or more of the higher sensory nerves appears in this leading metamere as a new formation, and thus a brain is constructed, or the vertebrate having a craniole containing a brain. This vertebrate is able to move with ease in every plane. We find lastly, in an appendix, experiments upon the semi-circular canals of the shark which have proved very suitable for experiment. According to the author, if the semi-circular canals are removed one after the other, the whole six can be taken away without the least disturbance of movement or equilibration being observed. If, however, with one canal the otoliths (Kalk konkremente) are removed, or if the vestibule is opened and the auditory nerves are stretched, disturbances of equilibration are observed without exception which as a rule take the form of rolling movements towards the injured side. Accordingly no function can be assigned to the semi-circular canals for the maintenance of equilibration. If

disturbances occur, they are forced movements provoked in the medulla, brought about by the stretching of the auditory nerves.

F. W. MOTT, M.D.

*Etudes Cliniques sur Les Maladies Mentales et Nerveuses.* 1 vol., 8vo, pp. 620. *Les Aliénés et Les Asiles D'Aliénés.* 1 vol., 8vo, pp. 560. Par le Dr. JULES FALRET. Paris: Baillière et Fils, 1890.

READERS of French medical literature, who are interested in mental diseases, will feel grateful to the distinguished President of the Medico-Psychological Society of Paris for collecting these portions of his writings, and presenting them in this accessible form.

M. Falret's prolonged and fruitful labours in the advancement of psychological medicine are so widely known, and his teachings are so largely incorporated in the mass of our present knowledge, that a lengthened detailed notice of the contents of these volumes is unnecessary.

In the first volume five articles on general paralysis occupy about a third of the pages. In the last of these articles (written in 1878) the author notes some of the changes which have taken place in relation to the alleged duration of the malady. At first stated to be six months, or a year, then limited to three years, it has ceased to be arbitrarily defined. He agrees with Douthett and Camille Lionet that the duration of general paralysis is longer in those who are descended from insane or neurotic ancestors than in patients who are free from hereditary taint. M. Falret is indisposed to assent to M. Baillarger's contention that the form of general paralysis, entitled by the latter *manie congestive*, is susceptible of permanent cure.

A very able, and exhaustive essay on the mental state of epileptics (written in 1860), with notes of several pertinent cases, occupies seventy pages, and has lost none of its interest or value from lapse of time. Following this is an almost equally lengthy paper on aphasia and allied disorders (1864), which is full of interesting matter.

An article on *Folie à Deux ou Folie Communiquée*,<sup>1</sup> written in

<sup>1</sup> The alternative title is the more accurate. A notorious instance of *folie à trois*, that many times has become public in metropolitan and certain provincial police reports during the last few years, instances the defect of the first.

conjunction with Lasègue in 1887, is an excellent monograph on the subject. In their summary the authors conclude that the communication of insanity is only possible in exceptional conditions, in which one of the individuals is the more active element; the delusions originate in him, and he gradually imposes them upon the second. The latter at first resists, then, little by little, submits to the pressure of his congener, and the delusions become common to both; they repeat them in the same terms, and in almost identical fashion. In order that this intellectual process be accomplished in two different minds it is essential that the individuals live for a long period in the same sphere, sharing the same mode of existence, the same interests, fears and hopes. Another essential condition is that the delusions shall possess a character of verisimilitude, that they are confined within the bounds of possibility, that they are based upon past occurrences or upon fears or hopes conceived for the future. This speciousness alone permits the paralogy of the one to be implanted in the mind of the other. Communicated insanity is more frequent in females than males. The chief therapeutic indication is separation of the affected individuals from each other: recovery may then take place, especially in the second, who usually is less profoundly deranged than the first.

The final paper in the book is on Circular Insanity—a form discovered by M. Falret's father and M. Baillarger in 1854. Written in 1879, the essay teaches that the melancholic phase of this malady is marked by an important general feature, viz., that the degree of the emotional depression of the simple melancholia is much more prominent than are delusions—if any exist. Another peculiarity of patients in this state is that, although they may be mute, immobile, and seemingly plunged in stupor, they observe and reflect much more than patients suffering from melancholia with stupor; in reality they see and hear all that goes on around them—a fact which they are apt to make known when they pass into the stage of excitement. The mania is chiefly characterised by hyper-activity of all the faculties, without predominant delusions; but in spite of their habitual gaiety and self-satisfaction, these patients are often the most malevolent, teasing, and mischievous of all lunatics. The symptoms of this phase usually are quite distinctive of *folie circulaire*; there are, however, cases of circular insanity in which the excitement may be much less pronounced, and others in which it may assume the form of an ordinary attack of mania. In the great majority of cases a lucid interval follows the melancholic stage: this interval may succeed each of the insane

periods, or it may be absent—in the latter event constituting *folie à formes alternées*.

The affection is essentially constitutional and hereditary, commences most often after puberty, without appreciable determining cause. Once fully established it lasts throughout life.

It is not sufficiently known (continues the author) that one frequently meets in families, and in society, persons who are not regarded as invalid, still less as insane, who pass their whole life, unknown to their *entourage*, in a successive rotation of periods of moderate excitement and of slightly marked melancholy, and who really are affected in a slight degree with *folie circulaire*. Such is its slightest degree—of necessity—only met with out of asylums.

Persons affected with circular insanity, the same as most lunatics suffering from hereditary insanity, decline much less intellectually, and even physically, than the majority of persons, and especially than other chronic lunatics.

Women are affected in much greater proportion than men. When a patient has had several well-marked attacks of circular insanity, one may predict that he will display that form of mental disease during the rest of his life.

The medico-legal aspects of the affection are amongst the most difficult in mental pathology, and can only be settled by attentive and complete clinical observation of the several varieties of the disease.

The Principles to be followed in the Classification of Mental Diseases (1860), the Semeiology of Cerebral Affections (1860), Catalepsy (1857), Physiological Theories of Epilepsy (1862), and Moral Insanity (1866), are the remaining contents of this volume, each subject being treated with M. Falret's characteristic lucidity and ability.

The second volume commences with a study (1862) of the insane colony at Gheel, "which has served as the starting point of all that has been done in the way of allowing greater liberty to the insane." To discover the origin of the colony it is necessary to go back to a legend of the seventh century which relates how, in the preceding century a young Irish girl called Dymphne, converted to Christianity, sought refuge with the priest at Gheel to escape from the incestuous passion of her father. The morbid Irishman found his daughter and slew her. Canonization followed; after which, as numerous lunatics were cured at St. Dymphne's tomb, the population came to regard her as the patroness of persons mentally deranged. In 1340 a church was erected in honour of St. Dymphne. Lunatics brought in pilgrim-

age to her shrine were cured in large number ; those unfortunates who were not restored by the intervention of the saint were provisionally deposited in an annexe to the church, and subsequently committed to the care of the nearest country people.

In the author's opinion the Gheel system is neither so good as its partisans claim nor so bad as its adversaries declare. For recent cases requiring active treatment and close attention it is especially defective : difficulty of treatment and surveillance ; the abandonment of the patient without sufficient control into the hands of the guardians ; the less satisfactory food, lodging and clothing than are provided in an asylum, count adversely. Altogether Gheel has more to gain by approaching the asylum system, than asylums have by imitating Gheel. The most important result that can be drawn from a visit to the colony is the general conviction that many chronic lunatics may without danger be accorded much greater liberty than one would have supposed.

M. Falret in the next two articles, written in 1863 and 1864, examines the various systems of public aid applicable to the insane. Amongst these is the medico-agricultural asylum at Leyme (department of Lot), which is intermediate, as regards mode of organization, between the Gheel colony and ordinary asylums. The nucleus of this asylum was an ancient abbey of Bernardine nuns. In 1835 a zealous brother of the Order *Saint Jean-de-Dieu* bought the abbey and 120 hectares of land for the purpose of establishing a lunatic asylum. Exorcism was to be the therapeutic agent, voluntary contributions were to provide ways and means. The worthy friar at the end of fifteen months was unable to pay the purchase money, and also having failed therapeutically he was forced to return the property into the vendor's hands, leaving his patients in a deplorable condition. In 1844 the establishment passed into the possession of a company, which gradually replaced most of the old buildings by new ones built in conformity with plans supplied by Parchappe.

The asylum has an isolated situation in the centre of an extensive and fertile valley, traversed by a high road and surrounded by wooded mountains. Its insane population in 1863 numbered 420, the majority being females. The buildings and terraces are enclosed by walls. At night the patients are locked in ; during the daytime this also is the case with those whom the physicians deem unfit to go out. The others are free to wander in all directions ; they can work if they choose ; can loiter or ramble, or take a *siesta* on the grass in the shade of the beech



trees. Several patients who are unwilling to work for the asylum, others who work irregularly, abuse their liberty by working for the neighbouring country people; the avaricious parsimony of the latter, however, minimizes the evil by causing them to enforce the patients' return to the asylum at meal-times. Excepting idiots and demented, the patients eat little or much, as pleases them. The principal means of repression is confinement to the wards. The camisole is employed when necessary, and the patient thus restrained is allowed to promenade with others. The single-room is the most severe corrective instrument. No coercion can be applied without medical order. The garden of two hectares is entirely cultivated by patients, under the superintendence of one gardener; much surplus produce is sold. Casualties and escapes are very infrequent.

M. Falret does not approve of thus abandoning patients to roam, with no other guarantee than the habit they have acquired of returning in the evening at the sound of the clock. Let us not fall back (he says), under pretext of giving fuller liberty to the insane, into the state of abandonment and vagabondage in which these patients were found in the old asylums, or at large, destitute of all rule and medical control. He thoroughly commends the boarding-out system, when safeguarded by medical supervision and direction.

"Foreign legislation affecting the insane, and the proposed reforms in the law of 1838," is the title of an article dated 1869, in which the author shews that while foreigners have perceived the value of, and imitated, French legislation, the adversaries of the law of 1838 now seek to retrograde. Instead of endeavouring to further the admission into asylums of recent cases, during the most hopeful period as concerns recovery, they strive to create obstacles to admission and to multiply the preliminary formalities, lest any sane person should be sequestered as insane. Of the reforms proposed by them the least objectionable is to require an additional medical certificate before the patient can be received into an asylum. But as the French law already requires three, is a fourth necessary? asks M. Falret. A second means is to cause the certifying physician to take an oath prior to his signing his certificate. This would either be a superfluous formality, or if seriously carried out, would deter many physicians from acting. Another proposal is that no lunatic should be placed in an asylum until after a magisterial enquiry. This would be placing a purely medical question in the hands of a magistrate not necessarily possessing any special competence. A still more objectionable



project is that of making a public examination before a special tribunal a compulsory antecedent to sequestration.

The author regards the surveillance of public and private asylums in France as inefficient. The agents charged with it certainly are very numerous, but on account of that multiplicity they count too much upon one another and their inspection, which is mostly optional, is too often perfunctory. He recommends expanding a section of the law, which gives to prefects the power to visit all insane inmates of asylums, so as to organize a permanent and adequate commission of inspection.

Perhaps the most important essay in the book is one written in 1876 on the "Legal responsibility of the Insane." To exemplify the progress accomplished in the laws of various countries relative to the responsibility of lunatics, M. Falret cites in detail (not with admiration) the history of English legislation affecting the insane. In France the matter is pretty clearly defined—insanity and irresponsibility are synonymous terms in the eyes of the magistrate or judge. M. Falret admits that there are circumstances in which the question of partial or total responsibility may be discussed—*e.g.* intermittence, incubation, convalescence; he also believes that in exceptional cases of apoplectic dementia, aphasia, alcoholism, &c., lucid intervals of sufficient duration to involve the return of legal responsibility do occur. In delirium tremens and alcoholic stupor the individual should be accounted not responsible; also in subacute alcoholism with hallucinations of sight and hearing, incessant fears with homicidal or suicidal tendencies, the person should be held totally irresponsible. The legal responsibility of epileptics is one of the most difficult questions of forensic medicine, and can only be decided after minute and exact observation of each particular case. It cannot be laid down as a principle that all epileptics are insane, especially that they are so at all moments of their lives, yet it would not be just to assert that *a priori*, an epileptic should be considered responsible for his acts, on the contrary, the presumption inclines the other way. So long as the psychic manifestations of the neurosis are limited to mere irritability of temper and tendency to quarrel, one cannot regard these persons, in the intervals between the fits, as veritably insane, nor exonerate them from all responsibility. All that one could possibly admit, and this only in extreme cases, would be a diminished responsibility: the circumstance might extenuate but not exonerate. Paul Zacchias, physician to Pope Innocent X. (1674) published most circumstantial details, and opinions in many respects remarkably con-

formable to modern doctrines, relative to the various forms of insanity in connection with civil and criminal law, even including partial insanity. He maintained, too arbitrarily, that every epileptic should be exempt from all responsibility for a violent act committed during three days before or after a fit.

The mental derangement described by the author as *petit mal intellectuel des épileptiques* is often difficult to diagnose; but, once determined, irresponsibility during the seizure is undoubted. The characters of this disorder are its rapid invasion and relatively short duration, a marked tendency to violent acts, a sudden termination. It presents in the same patient identical symptoms in all the attacks, and is followed by more or less complete oblivion of what has occurred during the attack.

M. Falret holds that insane persons whom the law would exempt for criminal acts, should also be deemed incapable of valid civil acts. A copious bibliography, up to the year 1875, is appended.

In the next two articles "Dangerous Lunatics" and "Special Asylums for Lunatics styled Criminal," are considered. The writer thereof makes known his abhorrence of the title *criminal lunatic*; the term is to him a "monstrosity." He has been very active in opposing the erection in France of special asylums for such patients; the only thing he would permit is the erection of small annexes for certain exceptional patients, who are particularly dangerous, or who have committed very notorious crimes.

A case of murder and arson, presenting much that is of medico-legal interest occupies nearly 100 pages. "Consanguinity," "Amnesia," "Aphasia," and "The functions of articulate language," each is the subject of a lengthy memoir. The volume concludes with a short account of the use of potassium bromide in epilepsy (1871). The mode of administering the drug was to gradually increase the dose from 1 gm. to 4 or 8 grms. daily, and maintain the maximum dose for a very long period. In about half the cases (Bicêtre inmates) no good resulted: of the remainder, a notable diminution in the number or the severity of the fits occurred in the majority, the rest were so greatly improved as to suggest cure.

We cordially hope that M. Falret will carry out his contemplated publication of the clinical lectures delivered by him at the School of the Faculty of Medicine and at the Salpêtrière.

E. BIRT.

*West Riding Asylum, Wakefield.*

---

*Des Méningites Microbiennes.* Par le Dr. E. ADENOT.  
Paris : Baillière et fils, 1890.

DR. E. ADENOT in the above-named paper has collected all the facts bearing on the vexed question of micro-organisms as ætiological factors in meningitis.

In the first chapter the author gives a brief but very complete *resumé* of the whole subject, whilst the three succeeding chapters are devoted to the enumeration and description of the micro-organisms met with in cases of meningitis.

In 1875, Klebs (*Arch. f. Experim. Pathol.*, Bd. iv.), found rounded diplococci, often arranged in chains, in the ventricles of the brain of patients who had succumbed after pneumonia. Eberth (*Deutsch. Arch. f. Klin. Med.*, Bd. xxix.) described diplococci in the subarachnoid liquid of patients who had died of the same disease. Leyden (*Centralblatt f. Klin. Med.*, 1883, No. 10) reported a case of bilateral otitis followed by perforation of the tympanum, cerebro-spinal meningitis and death, in which the cerebro-spinal liquid contained numerous cocci resembling those previously described by Klebs. Leichtenstern (*Deutsch. Med. Wochenschrift*, 1885) examined the exudation fluids in the meninges of nine patients who had died of cerebro-spinal meningitis and found them swarming with monococci and occasionally diplococci.

Sänger (*Archiv. f. Exp. Path. Bd.*, xx.), isolated the diplococcus of pneumonia from the meninges of patients who had died of meningitis secondary to pneumonia. The same micro-organism has been met with under similar circumstances by Fränkel (*Deutsch. Med. Wochenschrift*, No. 15, 1886); Foa and Bordoni Ufrezuzzi (*Deutsch. Med. Wochenschrift*, No. 15, 1886), and Weichselbaum (*Wiener Med. Jahrbücher*, 1886, and *Fortschritte D. Medizin*, Berlin, Nos. 18 and 19). Indeed, the latter obtained his first positive results in 1884, although he does not appear to have published them until 1886. Netter (*Arch. Gen. de Méd.* March, 1887) wrote an important paper on "Meningitis caused by pneumococci with or without pneumonia." The same author (*Bulletin Soc. Anat.*, 1886,) reported two cases of fibrinous pericarditis, cerebro-spinal meningitis and pleurisy caused by the presence of pneumococci, although no pneumonia was met with at the *post-mortem* examination. For other data concerning the presence of micrococci in meningitis, the papers by Goldsmith (*Centralblatt*, 1887), Pitmann (*Arch. f. Exp., Path. u. Pharmak.*, Bd. xxiv.), Hauser (*Münch. Med. Wochenschrift*, 1888), &c., may be consulted with advantage.

Bacilli have also been isolated from cases of meningitis. Indeed, the form of the disease most frequently met with, namely, tubercular meningitis, is undoubtedly due to the presence of the specific bacillus of tubercle. Neumann and Schæffer (*Virchow's Archiv.*, Bd. cix.) have described as occurring in cases of meningitis bacilli resembling in many, though not in all respects, the specific bacilli of typhoid fever. It appears to be highly probable, however, that the micro-organisms isolated by these authors were typhoid bacilli, as the slight differences of which these authors speak are not of much distinctive value. G. Roux (*Bullet. Médical*, 1888, and *Lyons Médical*, 1888), isolated from the meninges of one patient of purulent spinal perimeningitis, and of another patient who had died of sporadic cerebro-spinal meningitis, two kinds of hitherto unknown bacilli. Lastly, E. Adenot found in the meninges of woman bacilli resembling in many respects the micro-organisms of typhoid fever, although the patient did not present during life the symptoms of enteric fever and the *post-mortem* appearances were entirely negative.

In the other chapters the author discusses at great length the pathology of meningitis, the mode of penetration of the micro-organisms, &c., but this part contains no new facts and no original views.

Dr. Adenot's paper is undoubtedly a valuable addition to the literature of meningitis. In the first place, the references to medical literature are numerous and generally accurate, and in the second place, it is the first time that an attempt has been made to establish a scientific classification of meningitis. Nevertheless, it must be confessed that Dr. Adenot's work would have been of far greater practical value and scientific interest, had it contained a few experimental facts demonstrating the possibility of producing meningitis by the subdural inoculation of pure cultures of the various microbes enumerated in the paper.

M. ARMAND RUFFER, M.D.

---

*De l'atrophie cérébrale partielle d'origine périphérique.* Par le DOCTEUR A. SIBUT. Paris: Libraire, J. B. Baillière et fils, 1890.

IN this paper Dr. Sibut reports the *post-mortem* examination of a woman who died when 70 years old, and whose right lower limb had been completely paralysed during sixty-seven years, the

paralysis having been caused by an attack of unilateral infantile paralysis. The patient was quite unable to move the right lower limb, which was four centimetres shorter than the left. The patient ultimately died of pneumonia.

On examination of the brain *in situ*, "the observer notices on the *left* hemisphere in the region of the fissure of Rolando corresponding to the upper third of the ascending frontal and parietal convolutions, a fairly well-marked depression resembling the mark which the pressure of the thumb would leave on a soft substance. The meninges dip into the depression thus formed, but are not unduly adherent. The depression is oval in shape, begins at a distance of two centimetres from the median fissure, and is three centimetres long and two centimetres broad."

After hardening in 2 per cent. bichromate of potash solution, the left ascending frontal convolution measured 6 cm. 5 in length (right 7 cm. 5). It was 12 mm. broad in its width (right 12 mm.), 8 mm. in its narrowest part (right 12 mm.), and weighed 3 grammes (right 5 grammes). The atrophy began at a point situated at a distance of two centimetres from the median fissure and measured five centimetres in length.

The left ascending parietal convolution measured 6 cm. 8 in length (right 7 cm. 8), was 10 mm. broad in its widest (right 10 mm.), and 8 mm. in its narrowest part (right 9 mm.). The length of the same convolution on the left side varied between 11 mm. and 8 mm. (right 11 mm. and 10 mm.). The thickness of the grey matter on the border of the Rolandic fissure was 2 mm. 8 (right 3 mm.), and 3 mm. 2 along the post-Rolandic fissure (right 3 mm. 5). The depression above noted began at a point 2 cm. 5 distant from the median fissure and measured 3 cm. in length.

Microscopical examination of the spinal cord showed that the right anterior cornu in the lumbar region was greatly diminished in size; the ganglionic cells had almost completely disappeared in some places, and in other places were far smaller than on the opposite side. The walls of the vessels on the same side were considerably thickened; the connective tissue around was more abundant, and the author also found in the same situation numerous darkly-staining granular bodies. The antero-lateral tract on the left side was far smaller than on the opposite side, many medullary tubes being atrophied and containing no cylinder axes. "*The lesions were limited to the lumbar swelling and in no other regions did we find similar changes.*"

The author cut and compared sections of the extending frontal

and ascending parietal convolutions of both hemispheres. He states that "the large pyramidal cells (ascending frontal convolution) of the left are distinctly less numerous than those of the right convolution. In the layer of cells situated below the large pyramidal cells we almost always found a considerable diminution of cells, when compared with the right side. Lastly, in the different layers it was exceedingly difficult to find the large club-shaped cells which were exceedingly numerous on the right side." The nervous fibres were also diminished in numbers.

The morbid appearances present in the left ascending parietal convolutions exactly resembled those just described, except that they were far less marked. Dr. Sibut also draws attention to the fact that there were no other signs of inflammation.

The author has collected twenty-three cases in which changes in the brain followed on amputation or atrophy of one of the limbs through disease, and six cases in which no such central changes followed. It is, however, impossible to attach very much weight to the last six cases, as microscopical examinations do not appear to have been made in any of them. The work ends with a carefully-compiled bibliographical index, and the whole paper is a valuable contribution to the knowledge of localisations of function in the brain.

M. ARMAND RUFFER, M.D.

---

*Spinal Concussion.* By S. V. CLEVENGER, M.D. Philadelphia and London : F. A. Davis, 1889.

THIS is a large book, but one which contains peculiarly little information of value, a great part of its space being occupied by *verbatim* quotations from well-known sources, and the remainder being generally so badly written as to be hardly intelligible. In support of the last statement, and as some excuse for our having possibly misinterpreted the author, we may adduce a few instances of his style of composition. The full title of the work is "Spinal concussion: surgically considered as a cause of spinal injury, and neurologically restricted to a certain symptom group, for which is suggested the designation Erichsen's disease, as one form of the traumatic neuroses." If this mean anything, it means that "spinal concussion" is a "symptom group," in which case it can hardly be a "cause of spinal injury." Another sen-

tence, taken from the preface, illustrates equally well an obscurity of style which may possibly have been an important agent in destroying whatever value these pages may possess: we read "The author regrets the unavoidable polemics of parts of the book, though it is by controversy that the truth is often evolved from error; and his strong feeling against the political corruption in our country is explained by its opposition to all progress through the mediocrity and ignorance in most of our public institutions, scientific and medical—placed there by men of like character." Such grammar renders difficult both understanding and criticism of the author's views, but as "unsparing criticism is invited," we feel constrained to attempt its supply.

Omitting any reference to a brief "historical introduction," we find in our author's second chapter an analysis of Mr. Erichsen's work, which is chiefly remarkable as admitting frankly the heterogeneous nature of that surgeon's "cases," and which ends with a useless list of all sorts of symptoms which were at different times observed in these. This is followed by a chapter devoted to a criticism of Mr. Herbert Page, which is of so diffuse a nature that any reply would require much space, but as this criticism possesses no intrinsic importance, such reply may the more readily be omitted. We may, however, note that the author's main objection to Mr. Page appears to be that the latter accepts as reliable the *positive* evidence of old autopsies, but rejects merely *negative* statements, a position in which we venture to think that any surgeon will agree with the English writer.

The next eighty pages contain an almost undiluted series of quotations from various writers, Oppenheim receiving a translation which will be useful to those ignorant of German. An extraordinary hiatus in this portion of the work is the entire omission of the French school of neuro-pathologists, but we are led to believe that Dr. Clevenger is unfamiliar with their writings from the fact that he invariably spells Professor Charcot's name wrongly, and that he feels himself able to dispose of the great French neurologist by such a phrase as "the Charcôt (*sic*) fiasco."

Having read 117 pages of introductory matter, we were hoping to arrive at Dr. Clevenger's own views, but we find next a record of twenty personal and fifty-four quoted observations, followed, apparently as a sort of interlude, by a chapter on "Traumatic Insanity." Then we find an account of "the spinal column," which is intended to epitomise our knowledge of the anatomy, physiology and pathology of the spinal cord, which is badly put



together and which abounds in such loose statements as "destructive lesions of the sensory tracts of the cord have a tendency to ascend: those of the motor tracts similarly descend"; "Alterations in the meninges induce sudden pains and jactitations," &c.

After all this preparatory matter we at last arrive at the "Symptoms of Erichsen's Disease," a chapter which we may naturally expect to contain the kernel of the work. As a definition we find, printed in italics, that "Erichsen's disease is a group of mainly subjective symptoms, of a nervous and mental nature, sufficiently characteristic to enable it to be recognised as a traumatic neurosis, distinct from other traumatic neuroses, with which it may or may not be associated. The most common cause of Erichsen's disease is a concussion of the spinal column, including its contents and nearest appendages."

This introduction is followed by an enumeration of symptoms which includes nearly every possible form of perverted nervous action, but no attempt is made to correlate these symptoms or to show how they may be combined so as to constitute a case of "Erichsen's disease." We do, however, find a tabular statement of thirty-one groups of symptoms, and we finally learn that "the foundation, frame and roof of Erichsen's disease is the back pain and rigidity, the insomnia and emotional disturbance, subjective and unapparent to the untrained observer though they may be," and we learn further that "the roof, frame and foundation of the house may be out of sight, but they nevertheless exist; at least you can generally safely infer that they exist, even though unable to directly demonstrate the fact." Truly a remarkable disease! One whose results are as varied as the capabilities of medical language, but which essentially manifests itself by three "subjective" symptoms, these being, perhaps, unapparent to the untrained observer (that is, we presume, to the patient, who alone can perceive subjective symptoms), occasionally unapparent to any one. The latter variety suggests that it might be difficult to recognise this disease when it took the form of appearing without symptoms, but this is not so, for the symptoms exist whether they are there or not, or at least we can infer that they may exist. We believe that any impartial reader will accept our last quotation as the final *reductio ad absurdum* of Erichsen's disease.

We have not yet referred in any detail to the cases brought forward by Dr. Clevenger in support and illustration of his position, deeming it better to consider these as illuminated by the light which he himself throws upon them, but we wish now to discuss these cases somewhat fully, endeavouring to ascertain



what, if any, common features they present. The quoted cases are mostly well known, and we may confine our attention to those which are original.

These cases are twenty in number, but we may at once clear away several as being utterly useless as scientific material. Case II. is not described, but is headed "Probable Malingering." Case III. was that of a gentleman, who after a fall upon the feet for a distance of some three yards, gradually developed some vaguely described symptoms, which may have been the result of any one of several lesions. He died two years and a-half later of "Organic disease of brain and spinal cord and acute melancholia," a complication which certainly would appear serious. Case IV. is recorded in six lines, but appears to have been an instance of cerebral concussion. Case V. is a head injury followed by general paralysis of the insane; as the man had a pain in the back this is an instance of "Erichsen's disease." Case XI. is an utterly useless record. Case XIII. is probably an organic lesion of the spine, there being slight angular curvature with phosphatic urine, sluggish rectum and sexual feebleness; anæsthesia of the lower sacral nerves was apparently not looked for. Case XIV. is doubtless an organic brain injury due to a blow upon the right occiput. Case XV. appears to have taxed unduly even Dr. Clevenger's diagnostic skill; we may be pardoned for not venturing upon an opinion after reading his report. Case XVI. was "tossed by an engine;" she had "a medley of symptoms," among others "hyper-æsthesia and anæsthesia general (*sic*) with some paresis of the injured leg." The "paretic" leg had sustained a compound fracture of the tibia. This patient had pains in her back and is thus another instance of "Erichsen's disease." Case XVIII. was the result of a fall upon the spine from a height of a story and a half: pain and stiffness of the back with "numbness and formications," lasted for a fortnight. Surely not a remarkable result of so severe a contusion, nor one requiring a new nosology. Case XIX. is a severe case of general bruising with no remarkable symptoms. About Case XX. we learn only that he is a "damaged man," and had at some period or other a pain in his back.

We may thus narrow the limits of our study to the remaining eight cases, but must premise that the records are, as might be expected from our earlier remarks, not satisfactory, and they are disfigured by such remarks as "the other surgeon enjoys the reputation of being a great society intrigant, politician, and facile swearer for the side that engages him." "Several igno-

rant doctors testified on both sides." "Page would have reported his case as cured," &c. Such remarks are out of place in a professedly scientific text book, and upon a stranger their only effect is to lower his estimation of their writer.

Case I. is described as "A case of concussed spine, causing the typical nervous symptoms known as spinal concussion." The patient, a healthy man, aged 30, was thrown from the seat of a railway carriage by a collision; he was "shaken up," but remained on duty for eight days, and then gradually began to fail; after some months he "applied for his old place . . . but was refused, and *the same day* he took to his bed," where he remained for eighteen months preceding trial of his action against the railway company. At the end of this period his chief symptoms were:—emaciation, muscular weakness and wasting, dilatation and sluggishness of pupils, subnormal temperature, "apparently" universal hyperæsthesia, with regions of anæsthesia, photophobia, mental irritability, &c. Recovery after the trial was very slow and imperfect, there being long lasting weakness of volition, irritability, aching of the back with cervical and lumbar tenderness, muscular tremor, general hyperæsthesia of the skin and special sense organs, but anæsthesia and more marked weakness of the left side, Romberg's symptom, and slightly exaggerated knee-jerks. We should have described this as a very bad case of neurasthenia, with probably the characteristic symptoms of so-called hysterical hemi-anæsthesia. At least we find no evidence of a *sui generis* "Erichsen's disease."

Case VI. received a blow from falling pieces of stone, upon the left parietal region, and between the scapulæ. In the course of two years there gradually ensued loss of memory, irritability, sensory hyperæsthesia, tinnitus, anæsthesia of entire left side, and of the whole face and head, motor weakness of left side, tenderness of the spine between the scapulæ, &c. Eventually dementia supervened. We are told that "unilateral pareses are characteristic of spinal concussion, particularly with head blows." Case VII. is very similar, and also presented marked left hemi-anæsthesia and motor weakness. There was "spinal pain and rigidity." Case VIII. is that of a child, aged 9, who while skating, fell upon the buttocks, and subsequently underwent an extraordinary moral deterioration; in this case the "spinal" symptoms of pain, rigidity, &c., are not noted.

Case IX. is briefly reported. A man fell from a considerable height, was unconscious for half-an-hour, and then suffered from cramps and pains in the back, girdle pains in the lower lumbar

and cervical regions and some sensory troubles, of which the most wonderful is that he “‘sees stars if the weather is rainy,’ probably indicative of barometric pressure, increasing cerebral congestive tendency in optic as well as in other centres!” Case X. is an instance of right hemi-anæsthesia affecting limbs, trunk, tongue, &c. Case XII. is described as spinal concussion and myelitis. The record leads us to suppose that there was no organic injury but a functional anæsthesia of the left leg, with the commonly associated symptoms. Case XIV., the result of a blow upon the right occiput and upper spinal region, followed by the usual symptoms of cerebral concussion and then by right-sided paralysis (which included the sphincter iridis), is almost certainly an organic brain injury. Lastly, Case XVII. was the result of most severe shaking in a railway collision. Symptoms set in very gradually, and were those of general neurasthenia, with spinal pain and rigidity. From the wording of the report, we strongly suspect the *bonâ fides* of the patient, who curiously enough was, as are so many of the victims of spinal concussion, a post office employé.

We have endeavoured to study these cases conscientiously and without prejudice, and having regard to necessary limitations of space to place their outlines fairly before our readers, and we can only summarise them by saying that we find it utterly impossible to identify any common factor which will justify their being described as instances of a special disease; indeed, the sole bond of union between them appears to be that they were seen by Dr. Clevenger. We admit readily that there is yet much that is obscure in the clinical history and the pathology of traumatic neuroses, but we absolutely deny that these records have cleared up one single point, much less that they have proved the autonomy of Erichsen's disease as “one form of the traumatic neuroses.”

Finding Dr. Clevenger to be, as we believe, about twenty years behind the present day as regards his knowledge of the traumatic neuroses and therefore in a state of exceptionally great confusion we read his chapter upon pathology with but little hope of information. The result justified our expectations. The chapter, like its fellows, consists largely of long quotations (a comparatively easy method of making books) and gives us a large amount of information about the sympathetic system, which is neither new nor well digested, after which we learn that “Erichsen's disease” is due to a lesion of the pre-vertebral sympathetic chain. For this assumption we cannot find a trace of evidence. Autop-

sies, of course, there are more. That many of the symptoms of the traumatic neuroses are, or may be, due to disturbances of vaso-motor or trophic arrangements may be readily admitted, but there is at least as much evidence that the sympathetic ganglia are the seat of the essential lesion of say rheumatic fever, as that disease of these bodies is the underlying lesion of any traumatic neurosis.

We lay down Dr. Clevenger's book with no unkindly feelings towards its author. We have criticised one who himself does not shrink from "polemics," and we can only regret that we have failed to find any good thing in the book which is before us. We have willingly ignored its many minor faults, considering it necessary to refer only to those broad outlines which might exercise a pernicious effect upon the progress of our knowledge.

WILLIAM THORBURN.

## Abstracts of British and Foreign Journals.

**Kehr on a case on Cerebral Injury** (*Berl. Klin. Woch.*, No. 41, 1889).

The patient accidentally fell, his head coming in contact with a revolving circular saw, thus sustaining a wound 15 cm. long, parallel to the sagittal suture, 1 cm. to the left thereof, extending from 4 cm. in front of the coronal suture, to 1 cm. in front of the lambdoidal, incising the brain to a depth of 2 or 2½ cm. at the middle of the cut; and a wound on the right side of head, reaching backwards for 7 cm., from 1 cm. anterior to the coronal suture and ending in the sagittal, splintering and depressing the bone without penetrating it or injuring the superior longitudinal sinus. Loss of consciousness and paralysis of the right limbs and right side of face were complete: the left half of body was the seat of continuous epileptiform convulsions. The latter ceased as soon as the depressed bone was raised: consciousness returned a few hours later: aphasia with right hemiplegia remained.

From a consideration of the position and extent of the brain injury it was evident that the motor centres for the arm and leg were directly involved, the facial and speech centres could only be indirectly affected by pressure. The function of the right third nerve was intact. Muscular sense in the palsied limbs was impaired; not so the cutaneous sensibility. Cremasteric and abdominal reflex were diminished on the right side. Bladder and rectum functions undisturbed.

The facial palsy disappeared entirely in the course of four days; on the fifth day speech returned. After four-and-half weeks of complete akinesia a slight degree of power in the right lower limb was suddenly regained; three weeks later ambulation was performed without external aid. Volitional control of the upper limb commenced to be apparent seven weeks after the accident.

**Alt on Chloralamide** (*Ibid.* No. 36).

Experimenting with healthy adults engaged at manual labour, Dr. Alt found that 2 grammes of chloralamide given at three p.m.

produced no effect; 3 g. caused slight dizziness in one of the recipients three hours subsequent to taking the dose. No disagreeable after-effects followed the night's sleep. A 4 g. dose soon brought on vertigo and numbness: in one case great agitation and loquacity, in another case headache (chiefly occipital) nausea and tendency to stagger, resulted. The acme of the unpleasant symptoms was attained three hours after ingestion. After eleven hours' sleep slight hebetude and moderate occipital pain remained.

Sphygmograms taken every fifteen minutes shewed no alteration of the pulse. In patients also the sphygmograph has not shown any change in the pulse beyond such retardation as might be explained by the sleep—even when 4 g. or more was given. Temperature respirations and diuresis presented no striking alteration. To test the effect on digestion 2-3 g. of chloramide were administered with food: the contents of stomach three and-a-half hours later were normal and no subjective disorder had occurred.

Dr. Alt has found the drug a very useful hypnotic in many cases of nervous and general diseases, fairly constant in its action and usually unattended by any discomfort. Occasionally headache, dulness and vertigo have been complained of next morning, but there never has been any injurious action on the circulatory, respiratory or digestive functions.

In a case of delirium tremens, with great excitement and high temperature, 4 g., followed in two hours by a similar dose, produced nine hours' good sleep; on each of the next two days three 2 g. doses were given. Recovery was complete on the fourth day. Severe chorea in a boy was almost arrested by 1 g. thrice daily for five days.

**Hemiatrophia Facialis Progressiva.**—Professor VLADIMIR M. BEKHTEREFF, of Kazan, (*Mierzejewski's Vestnik Klinitcheskoi i Südebnói Psikhjatrii, &c.*, 1888, vol. i., p. 228) relates the fifth Russian (and about the eighty-first international) case of progressive facial hemiatrophy, referring to a slightly anæmic but otherwise healthy and well-developed girl, aged 4, offspring of a healthy family. In infancy she had been often and freely “soothed” with a poppy infusion. When about a year old, she had been intensely frightened on one occasion; shortly afterwards her mother noticed a livid spot, and about two months later a marked depression, just below the left zygomatic bone. When examined by the writer two and-a-half years or so

later, the child's left cheek was evidently flattened and covered with deep wrinkles and grooves, that gave to it an oldish look. The region corresponding to the anterior surface of the body of the upper jaw and to the maxillary process of the zygomatic bone, was not only flattened, but even somewhat depressed, the skin being extremely thinned, smooth, tightly stretched over the subjacent bone, bluish (from translucent blood-vessels), the bone distinctly atrophied. A similar state of things was also noticeable in the buccal region corresponding to the left half of the lower jaw. Besides a general atrophy in that region there was a fairly deep vertical groove, situated half centim. from the median line of the chin. This groove was lined by a perfectly smooth, scar-like and visibly pigmented skin. There was also marked wasting of the labial subcutaneous cellular tissue near the left oral angle and of the left half of the tongue. The faradic and galvanic reactions of the atrophic muscles were present, though distinctly weakened, the tactile and pathic sensibility intact. The atrophic process, therefore, was localised in the regions of the middle and inferior branches of the left trigeminus and was complicated with atrophy of the tongue. Analysing his case, Professor Bekhtereff points to (1) its rarity; (2) the relative rapidity of the development of the disease, and he accounts for this circumstance by the early age at which it made its appearance; (3) its causation. While in almost all the cases previously published the disease had been caused either by traumatic injuries of the head or face, or by exposure to cold, by epilepsy, measles, scarlet fever and whooping cough, in his patient—line in Mierzejewski Erlicki's case (*vide* below)—it made its appearance after a sudden and intense fright. Hence, mental shocks must be reckoned amongst the causes of facial atrophy. (4) The pathology of the affection. Having reviewed and criticised various theories proposed—such as that ascribing the disease to some lesion of the cervical sympathetic nerve, Bitot's and Landé's *Aphasie progressive*, the theory localising a primary lesion at the Gasserian and sphenopalatine ganglia; Virchow's hypothesis concerning a centrifugal disease of peripheral nerves, the nerve-central theory admitting a primary affection of the trophic regions of the medulla oblongata, Prof. Bekhtereff comes to the conclusion that Wendell's recent case (*Berl. Klin. Wochenschr.*, No. 19, 1888, with autopsy and microscopic examination of peripheral nerves) seems to establish the fact that facial atrophy is caused simply by a peripheral interstitial neuritis, analogous to that observed in cases of fractures, various acute diseases, &c.



In other words, Virchow's teaching appears to come nearest the truth.

The first Russian case was described by Professor J. P. Mierzejewski and A. F. Erlicki in the *Mierzejewski's Vestnik*, &c., 1883, vol. ii., p. 140. It is the case of a thin, but well-made married lady of 31, whose father had been a heavy drinker and whose brother was epileptic. She had been enjoying good health up to her 10th year, when she had a fright, followed in a few hours by an epileptic fit. The fits then occurred every two or three months at first; but subsequently two or three times a month. About a twelvemonth after the accident, the patient's aunt noticed that the left side of the girl's face had become smaller than the right one. About three years later a marked vertical groove appeared along the median line of the chin, while the wasting of the left facial half generally wasted more and more every year. When 25 years old she married, the first coïtus being immediately followed by a severe epileptic fit, which proved to be the last. From that time, however, she began to suffer from agonizing headaches and toothache, as well as from occasional attacks of a partial clouding of her senses, ushered by vague, but highly disagreeable sensations about her head, the attacks lasting one or two minutes. On examination, about twenty-one years after the accident, a striking difference between the facial sides was found, the left one being "extremely wasted, and producing an impression of its belonging to an old woman exhausted by some severe disease." The decrease in size of the side was dependent upon a considerable atrophy of the lower jaw and extreme wasting of the temporal, buccinator and masseter muscles; the anterior portion of the digastric muscle, the mylo-hyoid, internal pterygoid and tensor of the soft palate were also found to be markedly atrophic. The skin was marked with several yellowish spots and little wrinkles, and thinned over the temporal region, as well as just below the zygomatic bone and along the vertical and horizontal raris of the lower jaw. The sensation, temperature, electro-muscular reactions, and the vascular system were quite normal. The authors do not entertain any doubt whatever that they had to deal with a tropho-neurosis of a central origin. Since the atrophic phenomena were localized exclusively in the region of the third branch of the fifth nerve, and mainly referred to the motor apparatus, the writers think that there existed a lesion of the *locus ceruleus* or *substantia ferruginea*, situated in the lateral angle of the fourth ventricle and containing the motor nucleus and sensitive rootlets of the



trigeminus. According to their theory, a sudden mental shock (fright) caused a temporary disturbance of the cerebral circulation, sufficiently strong to give rise to an epileptic fit. The disturbance was probably associated with a slight hæmorrhage, or thrombosis of a minute blood-vessel in the region of the left nucleus of the trigeminus, which lesion was followed by consecutive, slowly-progressing, circumscribed, morbid changes of certain nerve-elements ("of special trophic nerve-cells") of the nucleus. The second and all subsequent epileptic fits might, according to these authors, have resulted from some secondary alterations, the latter giving rise to a very slowly advancing atrophic process about the corresponding side of the lady's face.

The second case was reported by Professor Vinogradoff, of Kazan, in the *Rüsskaia Meditzina*, No. 17, 1884, p. 388. It refers to a fairly strong, well made and nourished Jewish gold-miner, aged 27, of a healthy family. He had always been quite healthy up to his 20th year, when, without any apparent cause, he was attacked with alopecia over the *right* parietal bone; this leaving a permanently bald streak along the sagittal suture. Two years afterwards, he commenced to work as a gold miner, amidst Siberian *taigas* (marshes), where he was often exposed to severe winter cold, and suffered from muscular and articular rheumatism. When 23 years old he, on one occasion, rode on horseback across an open space, in inclement weather, his left side being all the time exposed to a violent and ice-cold wind. Shortly afterwards he began to experience pain and numbness, as well as muscular twitchings about the *left* half of his face and neck, whilst about two years later (in 1881), a steadily progressing wasting of the facial side set in, accompanied of late by local neuralgic pain, with muscular spasms occurring several times daily. On examination (in 1883) there was found an extreme emaciation of the *left* side of the face, depending upon the disappearance of the subcutaneous cellular tissue, and an intense atrophy of the temporal and masseter muscles; the skin being considerably thinned, here and there wrinkled and covered with desquamating brown patches. The left palpebral slit was narrowed, and the eye deeply sunken into the orbit, the tip of the nose deviating towards the right, the left palatine arch thinned. The left facial bones, however, were normal. On the *right* side, in the parietal region, there was seen a fairly deep depression, measuring 7 centimetres in length, and 4 in breadth, beginning from the lambdoidal suture, and passing obliquely forwards and outwards to the frontal bone; bifurcating there into

two narrow grooves descending along the outer aspect of the frontal bone down to the superciliary ridge. The skin covering the depression was quite bald and thinned, and the subjacent bone markedly atrophied. The man, therefore, was suffering from *atrophia facialis progressiva bilateralis*; when 20 years old, without any apparent cause for it, the right-sided hemiatrophy had made its appearance, the process involving the osseous tissue, beside soft structures; while at the age of 24, exposure to an intense cold had given rise to the left-sided wasting process, limited this time to the soft parts alone. Prof. Vinogradoff's case is only a third instance of a bilateral progressive facial atrophy (the other two belonging to Eulenburg and Wolff) and supplies an additional proof in favour of Virchow's thesis, according to which the osseous structures are involved only when the morbid process makes its appearance before the twentieth year of the patient's life.

The third case (from Professor Mierzejewski's clinic) is related by Dr. Ivan I. Rojdestvensky (*Proceedings of the St. Petersburg Society of Psychiatrists*, 1885, No. 6, p. 42). It is that of a well-nourished and normally built girl of 19, of a neuropathic merchant family, who when 15 years old, often suffered from headache, toothache, "rashes" and "ulcers" about the head and face; she had measles when 9 years old, and at the age of 12 some severe febrile disease of three months' duration. When between 13 and 15 years old, at the time of her first menstruation she suffered from hallucinations of sight and hearing; at 13, she first noticed that her right cheek had become more sensitive to cold than the left one, while, one year later, the cheek commenced to waste, slowly at first, but afterwards and up to the age of 16, rather rapidly. At the same time shooting pains about the infraorbital and mental foramina, and occasional numbness about the whole cheek were prominent symptoms. On examination, at the age of 19, the author found that, while her left cheek was ruddy and well-nourished, the right one was pale, emaciated, furrowed, as if by the depressions and folds of old age, and markedly diminished in size; the decrease being caused by an almost complete disappearance of the subcutaneous cellular tissue, and by a considerable atrophy of the lower jaw the muscles seemed to be unaltered. The skin presented two small whitish patches below the zygomatic bone, 4 cm. in front of the auricles, and was "as if adherent" to the subjacent bones; whilst the right angle of the mouth stood higher than the left one, and the right half of the lips, soft palate and tongue, covered with wrinkles and thinned.

The right nostril being dry and subject to excoriations, sensibility to touch and pain was normal, but the thermic one increased; faradisation and galvanisation produced a vivid pain about the infraorbital and mental foramina. The taste sensations of the right side of the tongue were considerably lowered, though tactile sensation in the same organ was intact. The patient complained of the right side of her oral cavity being always dry.

The fourth Russian case (also from Prof. Mierzejewski's clinic), is that of Dr. V. G. Dekhtereff's patient (*Mierzejewski's Vestnik*, 1886, vol. i., p. 97; and the *Proceedings of the St. Petersburg Society of Psychiatrists*, 1885, No. 6, p. 45), a rather lean, palish and nervous, but otherwise healthy, well-proportioned and well-developed seamstress, aged 13, the daughter of a neuropathic and alcoholic mother. She had been always healthy up to the age of  $11\frac{1}{2}$  years, when she once happened, when going to sleep, to knock her head against her iron bedstead. The accident left no visible traces; pain being also absent. But shortly afterwards her hair, near the spot which had been struck, began to fall out, while later on she suffered from left-sided headache and frequent bleeding from the left nostril. When examined by the writer,  $1\frac{1}{2}$  year after the accident, her left frontal region proved to be flattened and separated from the right one by a median vertical elevation, ascending from the globella. Close to the elevation and almost parallel with it there was a distinct groove, 1 cent. broad, which, starting from the globella, also ran up to the edge of her hair, where it passed into a wedge-shaped bald area measuring 11 cent. in length and 3.5 to 4.5 in breadth, and situated over the left frontal and parietal bones about  $1\frac{1}{2}$  cent. away from the median line. The bald area was covered with a glossy, scar-like skin, thinned, smooth and rosy in the anterior portion, and quite white, dense and somewhat uneven in the posterior one ("as if from periostitis"). The skin over the forehead was brownish, the pigmented area forming a vertical broad streak running from the orbital edge towards the bald place, while just below the latter there was a glossy, smooth, scar-like, but similar brownish circular patch,  $1\frac{1}{2}$  cent. in diameter. Within this frontal pigmented region, the subcutaneous cellular tissue was absent, and distinct atrophy of the frontal bone itself could be made out, the osseous surface being uneven and furrowed by depressions and grooves. On pressure, only the supraorbital notch (representing the supra-orbital nerve) proved to be tender. Several other abnormalities were detected. There was complete inhibition of perspiration

over the affected area; the left optic disc was pale and there was left-sided relative anosmia (to ammonia assafoetida, sulphuric ether), and she bled every week, or even oftener from the nostril. The other functions and organs were quite normal. Discussing the case, Dr. Dekhtereff arrives at the conclusion that first, it is a traumatic trophoneurosis, at present localised in the regions supplied by the supraorbital nerve; secondly, it is simply an instance of progressive facial hemiatrophy in an early stage—a “*hemiatrophia incompleta*,” which, in a couple of years, will probably transform into a “complete” one. As far as the localisation is concerned, Dr. Dekhtereff's case is yet a third of its kind, the first having been published by Romberg (1851), and the second by Dr. Karewsky (1883).

VALERIUS IDELSON, M.D. (Berne)

**On the Question of Changes that take place in the Nervous Centres under the Influence of Peripheral Irritation.** By Dr. IODOVSKY.

At present the medical world is greatly occupied with the question of nervous disease of a functional character. This question is full of interest as it gives rise to a host of questions, among which is the following one:—Have not these functional diseases a basis of organic change in the nervous centres as the result of hyper-excitation of the peripheral nerve fibres? The author's attention is arrested by this question, and he tries to solve it by means of experimental research. Dogs and rabbits were made use of for experiment. He irritated the following nerves:—the vagus, the sciatic, the intercostal and the great auricular. The author used electricity and traumatism as nerve irritants. The irritant action lasted from seven to twenty days.

The author found that the central portion of the irritated nerve remained intact, but grave changes were found in the ganglionic nerve cells, vacuolisation was noted, both central and peripheral coagulative neurosis, and degeneration of the peripheral nerve fibres. Besides which, there was in some cases infiltration of the ganglionic connective tissue, with round elements as well as congestion of ganglionic vessels.

The author admits that in consequence of prolonged peripheral irritation, impulses transmitted by the intact portion of nerve or central ganglion cause an excess of expenditure of vital force in the nerve cells of this ganglion, rendering it impossible for the supply to meet the demand, although the nutritive

matter is not lessened in quantity. As an outcome of this excess in expenditure of functional activity, we have a series of atrophic conditions followed by death of this cell. The lesion in the central cells resulting from peripheral irritations, unaccompanied by change of the central portion of the nerve, has been explained by Professor Vredensky's researches, who proved by experiment that when the nerve is in a normal condition it suffers no exhaustion; whilst on the other hand, the lesion in the intercostal ganglions, as a trophic centre, is by no means a slight matter, as far as the spinal cord is concerned, for a change takes place in the grey matter as well as in the white. (*Waller, Schiff, Buffalini and Rossè. Luiger, Bekhtereff, Rosenbach and Zeleritzky*).

Starting from all these data the author concludes that in the majority of so called functional diseases of the nervous system organic change is really present.

**On Hypnotism in Frogs.** By E. BERNADSKY. (*Archives de Psychiatrie de KOVALEVSKY*, 1889, 3).

According to Kircher's experience it has already been shown that animals as well as men can be hypnotised, that is to say, they submit to the influence of external causes, which brings about a change in their nervous system, and thus we have offered to us a picture of hypnotism.

We are well acquainted with many clinical agents which act on the nervous system; some have a stimulating action, others on the contrary, depress. What will be the influence of these clinical agents in the hypnotic condition of animals? Bernadsky studied the action of strychnine, thebaine and atropine on hypnotised frogs, and found that under the influence of strychnine and of thebaine the hypnotic state from being profound became light, and moreover, that the reflex sensibility of the hypnotised frogs increased.

The influence of atropine on the hypnotic state of the animal is totally different, it helps to produce the hypnotic sleep and makes it deep, thus bringing about results of quite a different nature to those produced by strychnine and thebaine. The hypnotic state is therefore a form of sleep, but nevertheless differs from true sleep. During the hypnotic state the brain excitability is raised, whilst during ordinary sleep it is weakened. On the other hand, the activity of the spinal cord is lowered during the hypnotic state.

**On the Influence on the Nervous System of Extirpation of the Thyroid Gland.** By Dr. S. AUTOKRATOFF.

Before publishing the results of his experiments on animals, the author gives an account of the historical development of the theory of the function of the thyroid gland as laid down by physiologists. Then comes an account of the traditional theory of the cachexia of myxœdema, and finally a full account of the author's experiments on animals. The animals who had been operated on after the second or third day after the extirpation of the gland exhibited a certain heaviness and want of activity and a marked disposition to remain lying down, their gait was a little unsteady and clumsy, their hind legs were spread wide apart, they could not sit down all at once, and they did so with difficulty, as though it cost them an effort to bend their legs. At times muscular rigidity was observed, their joints only bent with difficulty. Fibrillary contractions of the muscles took place, recalling Ewald's metaphor of a field of wheat which has been stirred by the wind. On the third day the tremor spread all over the body. The animals ate and drank sparingly and passed water frequently. The muscular tremor was soon followed by an epileptic attack with involuntary defecations, nystagmus, profuse salivation, and difficulty of breathing, which ended in death.

The tendon reflexes, at first exaggerated, were finally weakened. Death took place either very suddenly or with gradual renewal of the above described symptoms.

The galvanic excitability of the peripheral nerves in animals is usually increased after extirpation of the thyroid gland, but sometimes it is diminished. The electrical excitability of the psychomotor centres after the extirpation of the thyroid gland, when all the pathological symptoms have attained their maximum, was found to be exaggerated.

The author explains all the clinical symptoms which follow the extirpation of the thyroid gland in animals as an altered nutrition of the nervous system. The clinical picture most closely resembles tetanus in all its symptoms.

On making a microscopic examination of the brain and spinal cord of the animals who had died, the author found only a change in the grey matter, which had a cloudy appearance and infiltrated with leucocytes, the change in the cord was far more marked, and went so far as degeneration and vacuolisation of the nerve cells. The author concludes from his researches, that all this complex group of symptoms is due to suspended

function of the thyroid gland. As to the true character of this function of the thyroid gland it is at present an open question. The author believes that in the absence of the thyroid gland some unknown poison accumulates in the organism specially acting on the nervous system, and that, therefore, the function of the thyroid gland is to neutralise this hurtful material.

### **Influence of Hyperæmia on the Central Nervous System.**

By Dr. V. KOUSNEZOFF. 1889.

Following out the plan of Mendel and Furstner, the author produced cerebral hyperæmia, and found the following change take place in the central nervous system, namely, hypertrophy of the interstitial tissue, both in the cerebral vessels and also the neuroglia; the trabecula of the neuroglia was found thickened in several parts of the cord, and it appeared that not only was the number of the cells of the brain and spinal cord increased, but their size also. Degeneration of the nervous tissue had taken place, the protoplasm of the cells before their destruction underwent changes which finally brought about destruction and disappearance of the cell. The cells were more or less swollen, the protoplasm was pale, granular, at times vacuolated.

In other cases the cell presented a vitreous body that strongly refracted light, the processes disappeared, or were barely visible and degenerated, the nucleus which was at first visible finally disappeared. The nerve tubes also degenerated. At first the axis cylinders were hypertrophied, the swollen parts having the appearance of a string of beads from above, the swellings became vitreous, whilst others were granular and vacuolated. The whole granular mass seemed to melt from the periphery to the centre, and the cylinder of the axis gradually disappeared. The medullary sheath lost its contour and its primitive appearance; it seemed to melt and form again in separate drops. All these changes bore an inflammatory character. They took place in the following order:—At first the capillaries and the minute veins and arteries dilated here and there. Hæmorrhage took place, and very often plastic exudation occurred round the vessels and in the nerve tissues; furthermore a quantity of white globules was seen scattered about, tinged in part with red. Amongst the elements, serum, which had oozed from the capillary layer, made its appearance. Finally the change takes place which has been already described. The chief reason of this stasis is a check and hindrance to the circulation in the smaller vessels, and as Professor Paschoutin thinks is the cause of death.



**Procursive Epilepsy.**—By Professor P. KOWALEVSKY, *La Medicine*, No. 1, 1889.

Procursive epilepsy has recently attracted a good deal of attention, thanks to the labours of Bourneville, Ladame and Mairét. Professor Kowalevsky gives the history, symptomatology, etiology and progress of this affection, and adds a description of several cases which had come under his observation in his clinique for diseases of the nervous system. We give an epitome of a couple of his cases :—

The first patient was a Jew, 27 years of age, unmarried, who was a tailor by trade. His education was limited to deciphering letters so as to enable him to read with difficulty. The patient's father died of phthisis at the age of 32. The mother suffers from violent attacks of hysteria (?). No information as to any other of the patient's relatives. When he was 9 years of age the patient was taken from a little Jewish village to Kieff and apprenticed to a tailor.

In the following year a fire broke out in the neighbour's house. Everybody escaped out of doors, but the little apprentice was forgotten, and he awoke to find the flames of the house, which was on fire, licking the walls of his own little room. The child, mad with fright, lost consciousness for an hour. Subsequently he began to suffer from strange attacks, during which he took to running, regardless of anything that happened to him, taking no thought of why or where he ran. In about two or three minutes' time he was himself again, and was surprised to find himself in a neighbourhood more or less remote from his place of work, and where he had thrown his tools at the time of his seizure. When the attack was over he was worn out with fatigue. He was told that he always ran in a straight line, exclaiming "Oh! the fire, the fire, the fire!" He took no notice of anything that lay in his way whilst he ran, and when the attack was over he had no recollection of anything that had taken place. Those who witnessed the attack said that when it came on he turned pale, his eyes started out of his head, his whole expression was one of terror, and then he took to running. He never suffered from either hallucinations or delirium.

These attacks came at rare intervals at first, once in two or three months, but by degrees they became more frequent, and finally he had two or three attacks a day. Nevertheless there were times when, happily for the patient, he was free from attacks for a whole month, but this was not often the case. For the last five years the patient had attacks of momentary loss of consciousness, especially in summer.



When he came under observation he underwent the following treatment:—He took daily doses of bromide of ammonia, gram. iii., and iodide of potass. 0·2 gram., for two months' trial, and on alternate days the constant current was passed through his brain for five to seven seconds. He showed decided improvement. During the second week of treatment the attacks only came on about once in seven to fourteen days; a very marked improvement as compared with his state previous to treatment. Besides this he uttered no cry during the attack. He left Kharkoff, and did not return again.

The next patient was also a Jew, 26 years of age, unmarried. He was a printer's compositor by trade, and had received an elementary education. No information as to the health of the patient's father. The mother suffers from epileptic attacks. She had a large family of about twelve children, but the patient does not know how many have died, or what is the state of health of the survivors. The patient's native place is a small and poor little town, inhabited by Jews, and this is where all his family are living at present. When he was seven years of age the patient was taken to Vilna, and apprenticed to his uncle, a petty tradesman. He remembers that in the course of the same year he was excessively frightened and injured by a cow, and that after this fright his attacks of procursive epilepsy began. However, he is not quite sure whether the attacks followed immediately after the fright or some days after.

The attacks are characterised by a stamping of the feet without his shifting from his place. When the attack comes on he gets up suddenly, leaving the work he has in hand or a sentence unfinished, and then begins to stamp his feet. During the attacks he always mechanically unbuttons his dress and makes sounds like the lowing of a cow. The whole attack does not last longer than half a minute to a minute. The patient is unconscious during the attack, and has no recollection of anything that has happened when the attack is over.

These attacks at first came on once a fortnight, and went on in this way for ten years. After this period they became more frequent, even occurring as often as from eight to twelve times a day. The patient suddenly gets up, bends the knees slightly, inclines the body forward and stamps, lowing like a cow, and unbuttons his clothes.

His face is pale, his pupils are dilated, the pulse beats a little faster than usual, about 86 to 90 a minute. The attack does not last longer than a minute. It is interesting to note that the

patient feels the attack coming on for some seconds previously and in time to tell the medical man.

For the last five years he has not lost consciousness, during the attacks he wanders slightly. In the intervals between the attacks the patient is rather depressed, but no noteworthy change of intelligence has taken place.

PROFESSOR P. KOWALEVSKY, M.D. (Kharkoff)

**Reflex Epilepsy with Mental Change.** (*Zeitschrift f. Psych.* Bd. xlvii. p. 597.)

The patient whose case is here related had mental changes, with unequal pupils, and accompanied by epileptic fits coming on after the crushing of his hand. Fits were brought on by pressure on one finger. Bromides failed to relieve him, but after amputation of the finger, the fits did not return, although his mental condition remained unchanged.

**Tumour of the Corpus Callosum with Psychical Disturbance.** By Dr. DE LUTZENBERGER (*Neurolog. Centralbl.* No. 8, 1890).

The tumour of which the symptoms are described, was a large one, destroying the corpus callosum and the white matter of the parietal lobe and adjacent parts of the left hemisphere, and extending into the supramarginal and angular gyri. The tumour was a glioma with hæmorrhages in its substance.

The patient first shewed a certain amount of excitement and disturbance of vision. The later symptoms developed were weakness of memory and dulness of hearing, disturbance of articulation and unsteadiness in walking, with a tendency to fall backwards. He had later an apoplectiform attack, and subsequently remained more or less comatose, with exaggerated reflexes and contracted pupils.

**Nutrition in Hypnotism.** (*Compt. Rend.*, No. 15, vol. ii.) By MM. GILLES DE LA TOURETTE and H. CATHELINÉAU.

This research is on the same lines as that undertaken by the same investigators on nutrition in hysteria. The method used was the examination of the urinary excreta, and they find that in a period of hypnotism with its lethargic, cataleptic and somnambulistic stages, there is a diminution in the quantity of urine and in the amount of the solids, and that while this is well marked in the lethargic and somnambulistic stages it is still more marked in the cataleptic.

**Urine of Epileptics.** By CHARLES FERE (*Ibid.*)

The author undertook a research with the view of investigating the comparative toxicity of the urine of epileptics before and after a convulsion. MM. Deny et Chaupe in a similar investigation had been unable to establish any difference between the toxicity of urine under such circumstances, and of the urine under ordinary conditions. M. Féré, however, although not inclined to insist that his experiments settle the matter, finds that as a rule the urine first passed after a convulsion, injected into a rabbit, caused death without any convulsion, while the urine last passed before a convulsion rapidly produced death with strong convulsions.

In a subsequent number (No. 17), further experiments are detailed having the same bearing and similar results.

**The Superior Temporo-Sphenoidal Convolutions in one who was Deaf in the Left Ear.** By M. L. MANOUVRIER (*Bulletin de la Société de Psychologie Physiologique.* T. 5.)

This is a further contribution to the complete description of the brain of Bertillon. Since the publication of the description in the *Bulletin de la Société d'Anthropologie de Paris*, in 1878, the author accidentally learned that Bertillon was deaf in the left ear, so deaf as to be incapable of hearing what one sitting next him at table on his left-hand said. This deafness he had suffered from since infancy, and it was said to have been the result of an accident, the nature of which could not be accurately determined.

In the description of the brain already published, attention had been drawn to the large size of the left superior temporo-sphenoidal convolution, as compared with the right and this fact now attains great importance. The difference both in size and in the amount of convoluting present is remarkable, and much too great to be accidental, and this fact goes far to render it probable that in man hearing of one side is subserved by the superior temporo-sphenoidal convolution of the opposite side, as Ferrier has shown to be the case in apes.

A further examination of the brain in the light of this fresh knowledge shews some interesting points. Thus it is found that the part of Broca's convolution contiguous to the superior temporo-sphenoidal, is much better developed on the right side, *i.e.*, the deaf side of the brain, than on the left. As Bertillon was left-handed in his youth, it is suggested that so far as speech was concerned he was right-brained, and the difference in the development

of the auditory and speech centres on the right side is offered as an explanation of the difficulty which he found in expressing himself orally.

It is further pointed out that the angular gyrus is much more developed on the right side of the brain in this case than on the left and the explanation is offered that there is a hypertrophy of the part subserving the visual sense on that side to compensate for the deficiency in the auditory part. The ascending parietal convolution on the left side is found to have quite an extraordinary size, which the author accounts for by the close connection which must exist between the sensori-motor incitations connected with the limbs, and the sensori-ideal phenomena of auditory origin.

### **Concerning the Decussation of Fibres at the Optic Chiasma.**

By A. DELBRUCK (*Archiv. fur. Psychiatric, u. Nervenkrankh.*, 1890. Bd. xxi.)

At an autopsy the left optic nerve was found completely degenerated, the right partly. Both nerves, as well as the chiasma and optic tracts were cut in series, and stained by Weigert's method. The right uncrossed fibres alone were found normal. The uncrossed fibres were united in a bundle and running along the side of the nerve. In the chiasma and in the tract they were found to be mixed with the fibres which cross, although they were not scattered uniformly over the transverse section of the tract, but left free, at first the inner and lower quadrant, and later a gradually lessening marginal zone.

The author utilizes his facts to confute first of all the view that all the fibres cross, and in the second place the statement that the chiasma stops the degeneration.

After an examination of the literature bearing on the subject he comes to the conclusion that the disposition of the fibres which cross, and of those which do not, cannot as yet be accurately determined. It seems pretty certain, however, that the fibres which do not cross run in the nerve in a more or less definite bundle. In the tract, however, the rule seems to be that the direct fibres are mingled with those that cross.

### **Acute Disseminated Myelitis with Double Optic Neuritis.**

By CH. ACHARD and LOUIS GUNION (*Arch. de Med. Experim. et d'Anat. Patholog.*, 1889, No. 5).

This was the case of a young man of twenty-four, who, without any hereditary taint, and with no syphilitic history, became

blind in the course of six days, and sixteen days later developed paralysis of the lower limbs, with affection of the sphincters later and to a less extent affecting the upper limbs, and lastly respiration and deglutition. There was considerable sensory disturbance. After two months, spastic symptoms shewed themselves, but notwithstanding this, there was considerable improvement. Ten weeks after the commencement of the illness, sight was restored to a certain extent, sensibility became normal, and there was increased power in the limbs; three months later, however, the patient died of acute phthisis.

On subsequent examination three large patches of sclerotic change were found—one in the upper cervical region of the cord, another in the dorsal region, and a third in the optic nerves and tracts. In connection with these, secondary degeneration had taken place. In the patches were collections of large multinuclear cells both in the connective tissue and in the perivascular areas and these cells the authors regard as migrated leucocytes. Similar cells have been described by Küssner and Brosin in the *Archiv. f. Psych.*, Bd. xvii., also in a case of myelitis which they regarded as infective in origin. Achard and Gunion, however, do not venture to come to any conclusion as to the nature of their case.

**Morvan's Disease.** Clinical Lecture, by CHARCOT (*Prog. Méd.*, No. 11).

In this lecture Charcot draws special attention to a disease first described in 1883 by Dr. Morvan, of Brittany, of which several cases have since been recorded. The disease affects the upper, rarely the lower limbs, and is characterised by neuralgic pains, by wasting and anæsthesia, affecting the forearms and hands, and also by what is regarded as pathognomonic—the occurrence of whitlows on the fingers. As a rule, the whitlows appear last, so that as the anæsthesia is already established, they are not painful. It was the occurrence of a painless whitlow which first drew Morvan's attention to the disease. Sometimes, however, the first whitlow is painful, while those that occur subsequently are not. There is destruction of the phalanges, and on account of the anæsthesia, and the mutilation as well as muscular weakness which accompany it, an inability to work is produced. There are also present other trophic changes in the hands, such as cracks in the skin, deep ulcerations, coldness and lividity of the extremities, with profuse sweating. The nails also become shrunken and atrophied.

It will be recognised that in this disease there is some affinity to the symmetrical gangrene of Raynaud, and in the lecture the differential diagnosis of Morvan's disease from scleroderma, syringomyelia and leprosy is discussed. In the first named, the absence of anæsthesia and possibly the occurrence of the characteristic "masque sclérodémique" usually suffice to decide the question; in syringomyelia the muscular atrophy is of a different type, and thermic sensibility is said to be affected much more markedly than ordinary tactile sensibility, while in leprosy there will be the history of residence in some place where the disease is endemic, and there will be the change in the colour of the skin.

So far only one autopsy has been recorded, and in that case there was found to be degeneration in the posterior horns and also in the posterior columns of the cord.

JAMES TAYLOR.

### NOTE ON SUSPENSION.

MESSRS. RUSSELL AND TAYLOR, in their paper on "Suspension," published in the summer number of *BRAIN*, make several references to Motschutkovsky's cases. In commenting on Case No. IV. (in Motschutkovsky's paper) they remark that the patient appears to them to have suffered from "multiple neuritis" and not from "tabes."

Four cases are described fully in Motschutkovsky's paper and in the English translation made by myself. In the latter it will be observed that the diagnosis is given of Cases I., II. and III., while that of Case IV. is omitted. In Motschutkovsky's original paper and in the abstract of it (which appeared in the *Berliner Klinische Wochenschrift*, 1889, No. 25) Case No. IV. is described as an instance of "multiple neuritis." In the rough draft of my translation this diagnosis is also given. Hence I can only suppose that the omission is due to an error in transcribing or in printing.

This misadventure is, perhaps, not to be regretted, as it affords convincing proof that Motschutkovsky's cases are described sufficiently carefully and fully to enable the reader to form his own diagnosis.

S. W. SMITH, M.B.Ed.

Odessa, Sept., 1890,

# BRAIN.

PART IV., 1890.

## Original Articles.

### THE BI-POLAR CELLS OF THE SPINAL CORD AND THEIR CONNECTIONS.

BY FREDK. W. MOTT, M.D.

*(Lecturer on Physiology, Assistant Physician Charing Cross Hospital.)*

EXAMINATION of the spinal cord of man in reference to the connections of the cells of Clarke's column, the inter-medio-lateral, and the solitary cells of the posterior horn, is attended with many difficulties, which the anterior cornual cells do not offer. The latter, multipolar cells, give origin to the large fibres of the anterior roots, and are connected with similar cells above and below, and in the opposite anterior cornu. It would seem probable that their multipolar character is associated with these connections. The other cells of the cord are, for the most part, simply bi-polar. There is good reason to believe that nerve fibres bear a relation in size to the cells in which they originate. It is also probable that a nerve cell is larger when the nerve formed by its axis-cylinder process has a long course before it comes to its final termination. Hence the anterior cornual cells of the lumbar or cervical enlargements are larger than are met with elsewhere. A fibre, however, may branch after leaving the cell, as happens in the T-shaped fibre of the cells of the ganglion on the posterior root (Ranvier). This, however, does not apply to the fibres met with in the anterior roots, and it may be concluded that Gaskell<sup>1</sup> is

<sup>1</sup> 'Journal of Physiology,' vol. vii.



right in regarding small fibres as originating from small cells. He has shown that in the anterior roots throughout the dorsal region are numerous *fine medullated fibres* (leu-centric). These also are found in the second sacral root, and in the upper cervical region, and form the viscerovascular rami communicantes. If his first statement be accepted, that small fibres arise from small cells, the second statement that they originate from Clarke's column, which undoubtedly agrees in its distribution with this outflow of fine fibres by the anterior roots, cannot be accepted, because the cells of Clarke's vesicular column are by no means small, measuring where the column is well developed,  $90\ \mu$ , and even the smallest cells ( $40\ \mu$ ) are larger than the cells of two other groups, which he admitted might possibly be the source of fine fibres, and which I shall endeavour to show is much more probable. In fact, although text books state that Gaskell<sup>1</sup> has proved that these fine efferent fibres originate in Clarke's column, the whole evidence to support this hypothesis rests upon the assumption that because the cells of this posterior vesicular column are so placed in the cord as to agree in situation with the outflow of these fibres, therefore they give origin to them. No microscopical evidence tends to support this view. Occasionally fibres may be seen running from Clarke's column to the anterior cornua; especially have I noticed this in the mid-dorsal region. But the fibres cannot be traced into the anterior roots, and it may be that some of them pass into the anterior commissure as Bechterew states. Even this does not show that the fibres have originated from the cells of Clarke's column—in fact, they may be merely fibres

<sup>1</sup> Dr. Gaskell sums up his views of the functions of the various groups of the spinal cord:—(a) Cells of anterior horn = nucleus of efferent nerves to somatic muscles. (b) Large cells of lateral horn = nucleus of efferent nerves to striated splanchnic muscles. (c) Cells of Clarke's column = nucleus of anabolic (inhibitory) nerves to splanchnic glandular system and to muscles of viscera and vascular system. (d) Solitary cells of posterior horn = nucleus of motor nerves to muscles of viscera. (e) Small cells of lateral horn = nucleus of katabolic (motor) nerves to splanchnic glandular system and to muscles of vascular system.—“On the Relation between the Structure, Function, Distribution and Origin of the Cranial Nerves; together with a Theory of the Origin of the Nervous System of Vertebrata.” By W. H. Gaskell, M.D., F.R.S. ‘Journal of Physiology,’ vol. x.



passing out of the postero-external column on their way to the anterior horns.

Microscopical examination, however, shows beyond a doubt that fibres of the postero-external column, coming from the sacral and lumbar nerves, have their station in the lower part of Clarke's column. Physiological experiments in monkeys and pathological investigations in cases of injury and diseases of the roots of the cauda equina show degeneration of the fine nerve plexus around the cells of the posterior vesicular column.

The evidence of the lower connection of this group of cells will be first considered microscopically. If the cord of a foetus be examined in the lower dorsal region, sections stained by Weigert's method show bundles of fibres passing out of the postero-external column into the posterior horn like a brush (*vide* micro-photograph 1.), and limiting on the outside a group of cells lying in neuroglia substance. The cells are not larger than the adjacent solitary cells, and there is very little evidence of any fine plexus of nerve fibrillæ around them. The cells measure at this period about  $20\ \mu$  (average).

At successive periods of growth of the spinal cord, namely, four months, nine months, two years, and adult, transverse vertical sections showed the cells to be slowly increasing in size. At two years they are three times as large,  $60\ \mu$ , and in the adult even more than that, measuring on an average  $70\ \mu$  (*vide* micro-photograph 2). The latter measurement agrees pretty accurately with the average size determined by Sherrington.<sup>1</sup>

In the spinal cord of a child two years old I was fortunate enough to obtain a section showing fibres passing out of the postero-external column, breaking up into a fine brush of fibrillæ, with varicosities thereon around the cells of the column. (See fig. 2.)

Examinations of other sections from a very well-hardened adult spinal cord, made in a vertical direction, showed the

<sup>1</sup> Philosophical Trans. R. Society, vol. clxxxi., p. 33-48; 'Outlying Nerve Cells in the Mammalian Spinal Cord,' by C. S. Sherrington, M.A., M.B.

cells placed with their long axis vertical, lying in lymph spaces, surrounded by a spongy network of fine nerve fibrillæ. These could be traced across the lymph space into actual protoplasmic continuity with the cell. (See micro-photograph 3.)

It is possible that the cells stain a sepia colour by Weigert's<sup>1</sup> method, on account of the cell being covered with these delicate fibrils, the optical section of which give it its fine granular appearance. This fine plexus has long been known to be atrophied in cases of *tabes dorsalis*, and in injury of the roots of the cauda equina, associated with a sclerosis of the fibres of the postero-external column. The striking features of *tabes* which may be associated with this pathological change, are absence of the knee jerks, and loss of the guiding sensations, which normally operate in a reflex manner in the production of unconscious equilibration. Visceral disturbances are not at all uncommon, and Dr. Ross was the first to suggest that visceral crises and other disturbances were due to affection of the cells of Clarke's column.

It is possible, as suggested by Dr. Ferrier,<sup>2</sup> that along with tactile, common sensory, visual or labyrinthine impressions, other sensory impressions are correlated in the cerebellum, with the motor adjustments necessary for stability and equilibration. It may be that visceral or organic sensory impressions are represented in the cerebellum, but it is quite probable that other smaller cells in the immediate neighbourhood of Clarke's column, namely, the solitary cells, or the cells of the intermedio-lateral tract convey these organic sensations. Lissauer and Krauss, Oppenheim and Sæmmerling,<sup>3</sup> and others, have shown that the earliest change in locomotor ataxy is an atrophy of this fine nerve plexus. I described a case of injury to the roots of the cauda equina which showed this condition, together with a sclerosis of the fibres entering it from the

<sup>1</sup> Ct. Lenbossek, *Archiv. Microsk. Anat.*, v. xxxiii., 1889.

<sup>2</sup> *Functions of the Brain*.

<sup>3</sup> Krauss, *Zur Pathol. Anat. der Tabes Dorsalis*, *Neurol. Centralblatt*, Feb. 1st, 1885; Lissauer, *Archiv. für Psychiatrie u. Nervenkrankheiten*, 1885; Oppenheim and Sæmmerling, *Archiv. für Psychiatrie* vol. xx.

postero-external column.<sup>1</sup> The cells were somewhat shrunken, but there was no degeneration in the direct cerebellar tract. Cases of tabes have been mentioned to me in which the cells of Clarke's column were absent, but there was no degeneration in the cerebellar tract. This was due, as afterwards found at my suggestion, to the cells having tumbled out owing to the atrophy of the supporting fine plexus. For when the same cord was examined, after imbedding in celloidin, the cells were found to be as numerous as in normal sections.

With a view of determining the connection of the cells of Clarke's column with the fibres of the posterior roots, I made the following experiments:<sup>2</sup>—Unilateral section of the posterior roots of the cauda equina was made in two monkeys, the lower lumbar and sacral nerves being divided. The animals lost sensation in the lower limb of the same side, and there was absence of the knee-jerk. They were killed at the end of a month with chloroform. On microscopical examination of sections stained by Weigert's method the following degenerations are observed:—In the lower lumbar and upper sacral segments corresponding to the roots divided, there is complete degeneration of all the fibres entering the posterior horn, and a uni-lateral degeneration of the whole posterior column, except a small triangular area at the outer extremity of the posterior median fissure. Neither internal, central, nor external root fibres are seen, and there is a complete absence of the border zone of Lissauer. Moreover, an ascending bundle of small fibres seen at the outer part of the posterior horn of the uninjured side is quite absent. At the junction of the first and second lumbar segments the root fibres are seen undegenerated, and a triangular wedge of undegenerated fibres makes its appearance to the median side of the posterior horn. An examination of Clarke's column at this level shows an absence of the fibres which

<sup>1</sup> 'Journal of Anat. and Physiology,' vol. xxii., "On the Microscopical Examination of Clarke's Column in Man, the Monkey, and the Dog."

<sup>2</sup> These experiments were performed at the Brown Institution in the year 1888, by the kind permission of Professor Horsley.

pass from the postero-external column, and on the side of the lesion atrophy of the fine fibrillary plexus in which these fibres terminate. The number of cells is about equal on the two sides, but the difference in appearance of the fine fibrillary plexus was most marked in every section, notwithstanding the great number examined. The fibres which enter Clarke's column from the postero-external column may be divided into two sets, those which come from the outer division of the inner root fibres, and those which seem rather to limit Clarke's column than enter into the formation of the fine fibrillary network. Just above the lesion these fibres which run on the outside of the column, begin to appear, but still the fine nerve network around the cells is atrophied for some considerable distance above the lesion. In fact, it is not until we come to the seventh dorsal segment, where Clarke's column has become comparatively small, that the fine fibrillary plexus appears equal on the two sides. Coincident with this is an absence of all degeneration except in Goll's column; that is to say, not until the degenerated fibres have all disappeared from the postero-external column, does Clarke's column, as regards its fine plexus of fibrillæ appear the same on the two sides. The degeneration of Goll's column can be traced up to the post-pyramidal nucleus. The area of degeneration is smaller the higher it exists in the cord—probably due to the fibres themselves becoming smaller.

The degenerations are indicated in figs. 1 and 5. It seems probable to me that most of these large fibres which end in Clarke's column, and which degenerate when the lower roots of the cauda equina are divided, are supplied by the nerves of the lower extremity. These results accord with those of Tooth, with regard to the area of degeneration resulting from unilateral section of the roots of the cauda equina.<sup>1</sup>

Lissauer and Krauss, from their observations on the degeneration of the spinal cord in tabes, conclude that those fibres which lie most internally are most degenerated; and it is remarkable that the fibres which are most degenerated

<sup>1</sup> "Degenerations of the Spinal Cord," *Gulstonian Lectures*, 1890, Howard Tooth.

come from the lower roots, while those which form the outer group come from roots higher up.

Sherrington,<sup>1</sup> in an exhaustive account of the outlying nerve cells of the mammalian spinal cord, makes the following statement:—

“That the cells of Clarke’s column are connected on their proximal side with the fibres of the cerebellar tract admits of little doubt; as to their distal connections it may be said that two views are current. The one which has been supported by Mott is that the cells are connected with afferent fibres—fibres of the posterior root. The other (Gaskell, Hill) is that Clarke’s column is connected with the efferent fibres of anterior roots. Of these views the former, I think, derives support from the facts observed with regard to outlying cells in the posterior root zone of the cord. But it is difficult to believe that these outlying cells, although they appear equivalent to members of Clarke’s column, can be at all closely connected with any fibres in the anterior root. Indeed it appears more likely that not only the vesicular group of Clarke, and the outlying cells of the external posterior column, but also the cells appearing in or near the gelatinosa, all belong to the afferent system entering by the posterior root.”

Histology, physiological experiment and pathological observations all tend to prove that the cells of Clarke’s column are brought into relation with fibres of the postero-external column by means of the fine nerve network in which these fibres end.

We have now to consider the upper connection of Clarke’s column. As already stated, histological observations tend to show that the cells of Clarke’s column give off large fibres, which run upwards, slightly forward, and then outwards through the lateral column, to reach the direct cerebellar tract of the same side. Pathological observations strongly support this view, although Tooth<sup>1</sup> places the point of origin of the fibres in the direct cerebellar tract as somewhere between the ninth and eleventh dorsal, and states that it is probably higher in the monkey. He also concludes that the posterior vesicular column is in connection with fine fibres, because compression of the cord below the ninth dorsal (where the column is best

<sup>1</sup> *Loc. cit.*

developed) has, in a number of instances, not produced any degeneration in the direct cerebellar tract. He argues, therefore, that these cells cannot be the source of the fibres of this tract, but rather are connected with fine fibres which may ascend in the posterior median and antero-lateral. With regard to the former conclusion, neither pathology nor experiment supports it. I have lately recorded a case in which there is complete degeneration of the whole of Goll's column, with no degeneration elsewhere in the cord, Clarke's column being perfectly normal. Neither does his conclusion agree with the results of unilateral section of the posterior roots.<sup>1</sup>

Finally, Dr. Tooth (page 60) states that the "*true* direct cerebellar tract consists of large fibres, the antero-lateral principally of small ones. The direct cerebellar tract derives its fibres from the posterior roots, the antero-lateral from the grey matter of the cord—at any rate not from the posterior roots." I must confess that I cannot understand, if this be so, why no degeneration of the direct cerebellar tract results from section of the posterior roots of the cauda equina. The reason why degeneration of the direct cerebellar tract does not occur unless the compression is at the ninth dorsal or above it may probably be accounted for in this way:—the tract itself does not exist as a collection of fibres on the periphery of the cord until we reach the ninth dorsal, because the fibres which form it have to pass upwards, forwards, and outwards through the lateral column. A transverse section<sup>2</sup> of Clarke's column shows numbers of large fibres cut transversely. Moreover, the cells are placed, except in the lowest part, with the long axis vertical. These large fibres I believe to be the medullated axis cylinder processes of the large bi-polar cells. The very position of the posterior vesicular column in the centre of the cord would necessitate a severe crush in order to destroy the cells and their fibres. Whereas a much slighter compression, for example by a tumour,

<sup>1</sup> *International Journal of Medical Science*, January, 1891.

<sup>2</sup> *Vide* micro-photograph 2.

higher up, where these fibres have come to the surface, would more readily injure them. The evidence which supports the view that the cells of Clarke's column give off fibres which are continued upwards as the direct cerebellar tract is supported by the study of the development of this tract in the foetal spinal cord by Flechsig, Bechterew, and others. I have myself observed in the foetal spinal cord, passing out across the base of the posterior cornu, numbers of fibres which can be traced outwards through the front of the still unformed pyramidal tract. Vertical and transverse sections of the spinal cord in the lower dorsal region show large fibres passing out from the large bi-polar cells of this column, but they cannot be traced throughout the whole extent of their course, because they run upwards, forwards and outwards, and therefore, whether the sections be made transverse or vertical, their continuity must be severed. The fibres cannot, therefore, be traced directly from the cell into the direct cerebellar tract. With the appearance of the cells of Clarke's column in the lower dorsal region I have noticed large fibres scattered through the lateral column. To make more certain of this connection I made the following experiment. Unilateral section of the posterior-vesicular column was effected in a monkey at the level of the twelfth dorsal and first lumbar segments, and the animal was killed with chloroform after twenty days. A microscopical examination of the cord after hardening in bichromate of ammonia and staining by Weigert's method showed that the posterior column on the side of the lesion was almost completely destroyed; at the same time there was destruction of the grey matter on that side. (See micro-photograph 4.)

Microscopical examination of the sections with a high power showed, on the same side as the lesion, a complete atrophy of Clarke's column and disappearance of the nerve cells and fibrillary plexus at the level of the twelfth dorsal and first lumbar segments (*vide* micro-photo 5). At the ninth and tenth dorsal degenerated fibres were seen coursing out through the lateral column, and above this they were seen to reach the periphery; at the sixth dorsal the whole of the



degenerated fibres *had* reached the position of the direct cerebellar tract (*vide* micro-photo 6), and there were now no degenerated fibres seen in the cross pyramidal, thus coinciding with the experiment of Dr. Sherrington<sup>1</sup> upon descending degenerations. He found that a number of large fibres in the cross pyramidal tract of the lower dorsal region did not undergo degeneration; in all probability they were these fibres. Another fact which supports Dr. Sherrington's views is that the degenerated fibres in the direct cerebellar tract were all very large, but there were a number of smaller fibres mixed with them undegenerated, probably belonging to the cross pyramidal system. The degenerated fibres were traced up into the cervical region. In these were some few degenerated fibres on the other side of the cord, probably due to the lesion not being absolutely unilateral.

Two other experiments were made.<sup>2</sup> In one the unilateral section was made at the eighth dorsal. This produced only slight degeneration of the direct cerebellar tract, but there was considerable scattered degeneration in the antero-lateral. In another, unilateral injury of the postero-external column was produced about the level of the first and second lumbar, but the grey matter was not injured; in this case there was no degeneration of the direct cerebellar tract. I did not observe any visceral vascular disturbances in these animals. Of course the great difficulties attending such an experiment as the incision of Clarke's column are almost insuperable on account of the effects produced by injury of other portions of the cord; but the principal portion of the cord injured is the postero-external column, which has to be cut through in order to reach the vesicular column of Clarke. Section of the roots, however, has been shown to cause complete degeneration of the posterior column without any change in the direct cerebellar tract. It may therefore be reasonably concluded that *the cause of the degeneration of the fibres of the direct cerebellar tract is the destruction of the cells of Clarke's column.*

<sup>1</sup> 'Journal of Physiology,' vol. x., No. 5.

<sup>2</sup> The result of these experiments were made the subject of a communication to the Physiological Society, November, 1889. At the same time the microscopical specimens were exhibited.



Clarke's column extends from the first dorsal to about the second lumbar. It is best developed however in the lower dorsal region. A few somewhat similar cells are found in the sacral region, termed "Stilling's nucleus," and a few in the upper cervical region. Drs. Gaskell and Hill believe that the homologue of Clarke's column in the medulla is the nucleus of the spinal accessory and vagus.

The cells of these "nuclei" are spindle-shaped bi-polar cells—not unlike the cells of Clarke's column, only not so large. And as we know that the lateral horn contains similar cells, which moreover correspond in size, I should be inclined to believe that the larger bi-polar cells seen in vertical and transverse sections of the lateral horn in the dorsal region of the cord corresponded more closely with the cells of the vagus accessory nucleus. The homologue of Clarke's column in the medulla is probably the "cuneate nucleus," the cells of which resemble in shape and size the average-sized cells of Clarke's column. Upon this point Sherrington<sup>1</sup> and I concur.

Dr. Alexander Bruce<sup>2</sup> showed sections of the medulla of the foetus, which demonstrated that the upward continuation of the "cuneate nucleus" was "Deiter's nucleus," and the cells of this nucleus are in connection with the fibres coming from the semi-circular (deep) root of the auditory, the fibres terminating in this situation. The cells of Deiter's nucleus resemble in size and shape the large cells found in the lower part of Clarke's column, and measure  $60\mu$ — $90\mu$  in their long axis. Schwalbe states that they are multipolar, and gives the measurement in their long axis as  $60\mu$ — $100\mu$ . When these cells first begin to appear in the ascending root of the auditory nerve they are mingled with smaller bi-polar cells belonging to the cuneate nucleus. The cells of Deiter's nucleus do not appear to me to be more multipolar in their character than many of the cells in the lower end of Clarke's column (*vide* micro-photo. 2).

If these cells, which closely resemble the large cells of Clarke's column, are connected with the deep fibres of the audi-

<sup>1</sup> *Loc. cit.*

<sup>2</sup> Meeting of the Physiological Society at Edinburgh, July, 1890.

tory nerve coming from the semi-circular canal, and serve as a station for the impulses transmitted from the semi-circular canals to the cerebellum, we may consider that impulses connected with equilibration are brought from the skin, joints, and tendons by fibres of the postero-external column to the cells of Clarke's column from the lower extremities, and conveyed thence to the cerebellum by the direct cerebellar tract. Impulses also may be conceived to come from similar situations in the upper portions of the body and to pass to the "cuneate nucleus," of which the cells serve as a station for impulses through the restiform body.

Organic sensations from the viscera may also be transmitted, as suggested by Dr. Ferrier, through the cells of Clarke's column to the cerebellum. All the evidence certainly goes to prove that these cells serve as a station for afferent impulses between the peripheral nervous system and the cerebellum. Only one histological fact of a doubtful nature shows that the cells of this column may be connected with the cells of the anterior horn.

There are two other groups of cells found in the spinal cord, which in shape and form differ essentially from the anterior cornual cells. These have been designated by Schwalbe the *intermedio-lateral* and *solitary*. The former are found throughout the dorsal region. The cells are bipolar, and in vertical sections they are often seen to exist as little groups or nests of vesicular cells, from eight to twelve in number. These cells measure on an average  $30\mu$ , have their long axis placed vertically, obliquely or horizontally, and closely resemble the smaller cells of Clarke's column. In transverse sections, as also in vertical sections, may be recognised not merely the cells previously described, but also two other sets of cells of a broad spindle shape, placed usually with their long axis horizontal to the transverse section. The small ones, average size  $20\mu$ , closely resemble in form and size, and are probably identical with, the so-called solitary cells. This appears to be also the opinion of Sherrington. The larger measure  $25\mu$ - $30\mu$ , and are probably identical with the cells placed with their axis oblique or vertical. The small spindle-shaped cells attain their full size, or nearly so at birth.

As in Clarke's column, so in this tract we find many cells lying out in the lateral column. The cause of this, together with the mingling of cells of different forms to such an extent, is probably due to the growth inwards of the fibres of the posterior roots, splitting up the cells of the grey matter, which lie posteriorly and laterally to the central canal of the spinal cord during its development in embryo, and also in the early years of post embryonic life, when vertical commissural connections of the cells at different levels have to be established, together with the growth downwards of the pyramidal tracts.

These fibres, both before and after birth, cut off small portions of grey matter, and so the cells become isolated and outlying. I have never seen them lying in the still undeveloped pyramidal tract of the fœtus. I do not make any positive assertion concerning these outlying cells, as I am at present engaged in investigating the matter.

Examination of a series of sections of the spinal cord of a two-years-old child for the determination of the position of the small ovate and spindle-shaped cells situated in the lateral horns:—

1st dorsal.—A few cells.

2nd „ Very numerous ovate and spindle-shaped cells; many placed long axis vertically, 20-30 each side.

3rd „ Ditto, 11-15 each side.

4th „ „ „ „

5th „ Lateral horn well developed; cells, large and small, 14-16 each side.

6th „ Well marked, 15-20 each side.

7th „ „ „ 12-15 „

8th „ „ „ 15-20 „

9th „ Numerous cells „ „

10th „ „ „ 10-15 „

11th „ Not so numerous.

12th „ „ „

1st and 2nd lumbar.—Some cells.

At the second sacral, as shown in fig. 4, some small ovate bi-polar cells were discovered in the sections. Too much reliance, however, cannot be placed upon this fact, because a large number of sections were examined in order to see if cells existed in this region which could, from their size and simple form, be associated with the small fibres which Gaskell has shown to emerge from the spinal cord in this region. The medium-sized bi-polar cells found

in the intermedio-lateral tract correspond in form and size with the cells of the vagus nucleus, and it may be considered that the smaller cells give origin to the fine fibres which pass out of the anterior roots throughout the dorsal region. This certainly seems much more probable than that they should come from the cells of Clarke's column. Throughout the dorsal region at the base of the anterior horn are found numerous small angular and spindle cells. These are found also in the upper cervical region, and probably are connected with fine efferent fibres.

Fine root fibres enter the posterior cornu on the outside, although they cannot be traced directly into the small bi-polar cells found at the base of the posterior cornu, and in the intermedio-lateral tract it is quite possible and probable that these small fibres find their station in these small cells, which are placed, with their long axis, horizontally, so that we might consider one pole of the cell to be connected (though not directly) with an organic sensory fibre, the other pole being continued forwards and outwards as a fine efferent fibre. Fine fibres certainly can be seen in vertical sections passing out through the lateral column, as represented in the diagram. Owing, however, to the cells being usually cut across it is difficult to trace the fibres any distance, because they tend to run forwards and outwards (*vide* fig. 3). In vertical sections of the lower dorsal regions large coarse fibres can be seen running across the lateral column. These are probably going to the direct cerebellar tract.

With regard to the larger cells resembling those of Clarke's column found in the intermedio lateral tract, with their long axis placed vertically, it is possible that these may be connected with fibres of the ascending antero-lateral. I think that the inter-medio lateral probably contains cells belonging to different systems, those placed vertically being connected with an ascending set of fibres, antero-lateral, those placed horizontally with efferent root fibres.<sup>1</sup>

Moreover the development of the spinal cord supports the view that the intermedio-lateral tract gives origin to the splanchnic efferent fibres of the anterior roots. He has shown that there are two kinds of efferent fibres, somatic or ventral, and lateral or splanchnic, and these take origin from entirely different parts of the basal lamina of the neural tube. The former originating from groups of neuroblasts, near the middle line, the latter near the junction of the basal with the

<sup>1</sup> Bechterew, '*Über den Nerven Wurzeln*,' *Archiv. für Anatomie*, 1887.

alar lamina. In connection with the origin of the cranial efferent nerves, Schäfer states that :—

“They are disposed in two longitudinal series, one of them comprising the nuclei of origin of the somatic efferent nerves of Gaskell, which correspond with the largest fibres of a typical anterior spinal root, and the series of nuclei is a continuation of the cell column of the anterior horn. The nuclei of this series are the 12th, the 6th, 4th, and 3rd. The other series comprises the nuclei of origin of the splanchnics, efferent nerves of Gaskell, which correspond with the medium sized and smaller fibres of a typical anterior spinal root, *and the series is a continuation mainly of the cells of the lateral cornu of the intermedio lateral tract*, and partly perhaps of the cells of the base of the posterior horn. The nuclei of this series are those of the spinal accessory, those of the efferent fibres of the vagus, glossopharyngeal, facial and motor nucleus of the 5th.”—QUAIN, vol. i, part 1.

Lying in the grey matter of the base of the posterior cornua, especially on the outer side, are numbers of bi-polar cells of a spindle shape placed with long axis horizontal, and with processes directed forwards and backwards—the so-called *solitary cells*.

In conclusion, although many writers have stated that Dr. Gaskell has *proved* that small efferent fibres of the anterior roots of the dorsal region are connected with the cells of Clarke's column, it is as well to state that this eminent authority did not exclude the possibility that they originated from cells of the inter-medio lateral or the solitary, which I have endeavoured to show is much more probable. Dr. Gaskell was under the impression that the cells of Clarke's column are small—hence the inference that small fibres are probably connected with small cells; and inasmuch as the distribution of Clarke's column corresponds to the outflow of fine fibres, it was natural that he should have leant to the view that Clarke's column was their source rather than the other groups mentioned by him. Gaskell<sup>1</sup> has recently referred to the cells of Clarke's column as divisible into two groups of different sized cells. Sherrington, in a footnote concerning this point, makes the following statement :—“I must confess, however, I have not been able to convince myself of the division of the cells

<sup>1</sup> ‘Journal of Physiology,’ vol. x., p. 157, 1889.

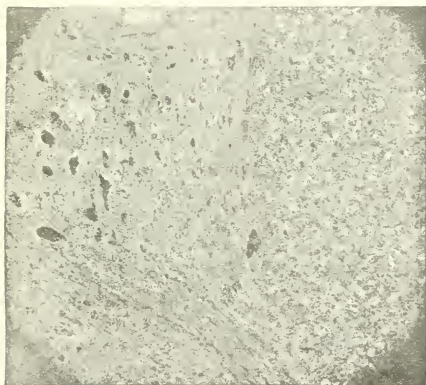
into two groups, nor do I interpret the observations of Mott<sup>1</sup> to be in support of the division of cells into two groups." I think I can explain this discrepancy.

Schwalbe<sup>1</sup> has pointed out in connection with the anterior cornual cells, that where a column is well developed the cells are larger—therefore the cells of the lower end of Clarke's column are larger. Moreover, owing to their connections with the fibres of the postero-external column on the one hand, as shown in fig. 2, and on the other hand with the fibres of the direct cerebellar tract, the cells are placed not with their long axis vertical, but either obliquely or horizontally, so that they appear in transverse sections much larger here than higher up. Consequently in transverse sections of the lower end of the column one sees the long axis of the cell, whereas higher up one sees the vertically-placed bi-polar cell cut transversely, and, therefore, only the narrower transverse axis is measured. I also stated that in the postero-external column were two sets of fibres, large and comparatively small, as shown in micro-photo. 2. The former I believed to be connected with the larger cells of Clarke's column, the latter with the smaller. This may be so, but still the impulses passing through the cells would be afferent. From experimental observations I am more inclined to believe that these smaller fibres belong to Goll's column, being on their way there through the postero-external column. This much may certainly be said, that the smallest cells I have seen in Clarke's column, under any circumstances, have been larger than the largest cells found in the other two groups. This fact, when coupled with the fact that at birth the cells of Clarke's column are only one-third of the size of the cells at two years, strongly suggests that they are not connected with fine efferent fibres having an organic function such as the innervation of the visceral-vascular system.

Moreover, the whole of the cells of the spinal cord would be absorbed for efferent fibres, according to Dr. Gaskell's theory, leaving no cells for two distinct afferent tracts to the brain, viz., the direct cerebellar and antero-lateral, which have been shown not to have their origin in the ganglion cells of the posterior root.

<sup>1</sup> *Loc. cit.*

TO ILLUSTRATE DR. MOTT'S PAPER ON THE  
BI-POLAR CELLS OF THE SPINAL CORD AND  
THEIR CONNECTIONS.



MICRO-PHOTO. I.

Transverse section of foetal spinal cord, lower dorsal region, shewing cells of  
Clarke's column; magnified 150 diameters.



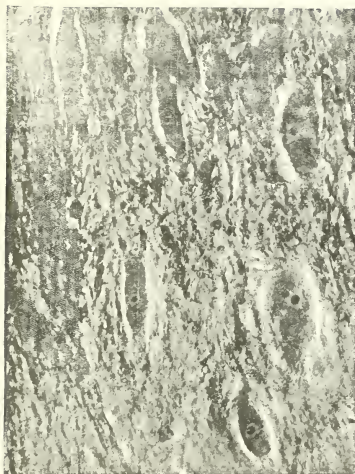




MICRO-PHOTO. II.

Transverse section of Clarke's column in the lower dorsal region (11th-12th segment), showing many large cells; and, in the fibrillary net work, large fibres are seen cut transversely. Magnified 250 diameters.





MICRO-PHOTO. III.

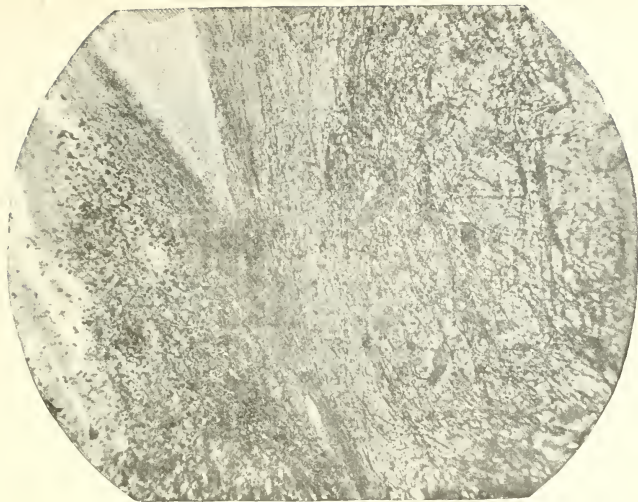
Vertical section of Clarke's column shewing the large bi-polar cells lying in a spongy network of nerve fibrillae. In the original photograph the fine fibrils can be seen passing across the lymph space to join the cell. Magnified 250 diameters.



MICRO-PHOTO. IV.

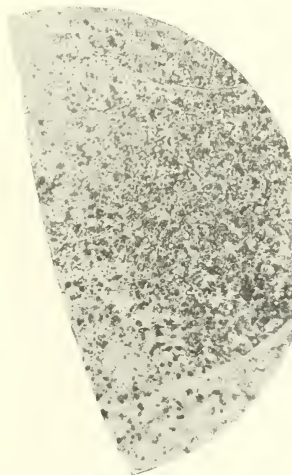
Spinal cord of monkey at 12th dorsal. The seat of the lesion is seen on the left side. Destruction of nearly the whole of the grey matter on that side, and shrinking of left half of the spinal cord. Magnified 10 diameters.





MICRO-PHOTO. V.

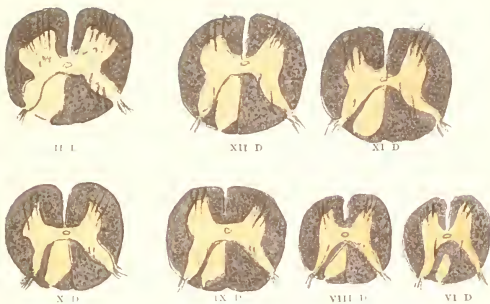
Clarke's column from the previous section, as seen under a magnification of 150 diameters. On the right hand side of the central canal are seen the cells lying in a fibrillary network; on the left, disappearance of the cells and atrophy of the network.



MICRO-PHOTO. VI.

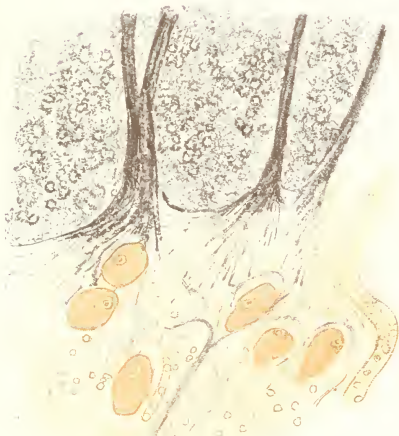
Portion of the lateral column in the mid-dorsal region on the side of the injury, showing the degenerated fibres, stained by Weigert's method, after hardening in celloidin. Nearly all the degenerated fibres are seen at the periphery in the direct cerebellar tract. Magnified 100 diameters.





1.—Unilateral section of the roots of the Cauda Equina in the Monkey.

Mag. 4 diameters



II.—Clarke's Column human spinal cord, 12th dorsal. Fibres from postero-external column entering and terminating in a brush of fine varicose fibrils around the cells which are for the most part cut across obliquely.—Mag. 250.





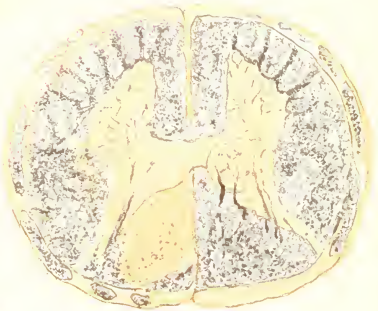


III.—Vertical section of lateral column and intermedio-lateral tract mid dorsal region, 2 yr. human spinal cord.

Mag. 250 diameters.

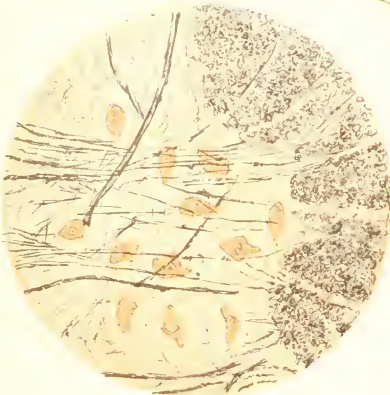
V.—Spinal cord of Monkey at level of 12th dorsal. Degeneration of postero-external column on left side as a result of section of 3d, 4th, and 5th lumbar and 1st and 2d sacral. Atrophy of nerve plexus around cells of Clarke's column on the same side.

Mag. 10 diameters.



IV.—Cells of intermedio-lateral seen in transverse section of 2d sacral.

Mag. 250 diameters.





# THE EFFECT OF MOVEMENTS OF THE HUMAN BODY ON THE SIZE OF THE SPINAL CANAL.

BY R. W. REID, M.D., F.R.C.S.

*Professor of Anatomy in the University of Aberdeen; and*

CH. S. SHERRINGTON, M.A., M.B.,

*Fellow of Caius College, Cambridge; Lecturer on Physiology in the School of St. Thomas's Hospital.*

*(From the Physiological Laboratory, St. Thomas's Hospital, London.)*

OUR object in making the following short series of experiments has been to test the possibility of an alteration taking place in the capacity of the cerebro-spinal canal during the performance of such movements as can, under ordinary or extraordinary circumstances, occur in the jointed walls of that canal between the bones composing the cranio-vertebra axis. A few preliminary observations fully sufficed to show us that a change of capacity of the canal does actually take place, although only to a slight extent. These detectable but small alterations it seemed highly desirable to measure, and obtain permanent records of. The following plan of experiment was therefore devised and in its ultimate form found successful.

The cadaver, the subject of observation, was suspended from the horizontal arm of a strong vertical stand by means of an iron coronet which encircled the skull and held it by four sharp-pointed thumbscrews, screwed into, but not perforating, the calvaria. An additional precaution against the slipping downward of the cadaver was obtained by inserting into the external auditory meatus the ends of a scissors-shaped clip (B). This clip was attached to a rope running over two pulleys at the top of the vertical support. A horizontal iron bar (C) which could be adjusted at any desired height on the vertical stand was employed to act as a fulcrum against which the body could be bent.

The procedure of experiment was as follows. The scalp over the vertex having been removed, a trepan hole measuring 35 mm. across was made in the highest part of the skull, close to one side of the superior longitudinal sinus. The dura mater was thoroughly removed to an extent corresponding with the trepan hole. We then generally found that the brain was separated from the parietes of the cavity sufficiently to admit the handle of a scalpel between it and the overlying dura mater. Water was then poured into the subdural space sufficient to fill up the trepan hole. The water introduced tended to disappear, often rapidly, sinking in level into the subdural space. Especially was this so with the first few ounces poured in; after a certain amount had been introduced the sinking became less rapid. When the trepan hole was quite filled with water a small glass tube (D) surrounded by an india-rubber ring was inserted with as little delay as possible into the hole, and was arranged to fit into it in a perfectly water-tight fashion. Over the end of the glass tube had been previously drawn a thin membrane which floated on the water filling the subdural space; this membrane was made sufficiently redundant to allow it a considerable range of movement on the water. The glass capsule itself was filled with water and put into communication by flexible water-tight joints, with a length of glass tubing almost horizontally placed (E). The farther end of this tubing was the lower, and was placed on the same level as the trepan hole in the skull. In the figure the inclination of this tube is represented as greater than it really was. Finally, this almost horizontal tube and the connections intervening between it and the vertical glass capsule were all completely filled with water.

Beneath the free end of the tube the scale-pan of a sensitive balance was placed in such a way that it received any drops of fluid ejected from the tube. To the other end of the beam of the balance was attached a thread, which passed vertically downwards, and after running round a light pulley hung, carrying a little weight and armed with a small pen arranged for writing upon the blackened surface of a recording drum. The amount of water received by the scale-pan

was represented by the height of the line marked by the recording pen. The scale-pan naturally underwent movement along the arc of a circle; the intervening pulley round which the thread passed converted the movement of the thread into a rectilinear one; in order, therefore, to estimate the amount of water in the pan the drum had before each set of observations to be experimentally graduated. In each observation the measurement recorded is that of the *maximum* displacement obtained in a series of repetitions of the particular movement.

I. *May 8, 1889.*—Cadaver of male child, æt. four years, weight 24 lbs. 10 oz. Cause of death, diphtheria. Trephined at vertex, to left of median line. Head fixed in the coronet in such a way that no part is higher than the median edge of the trepan-hole.

Body hanging freely from the head:—On raising the trunk by the pelvis, thus taking off the extension of the spinal column due to the weight of the trunk and limbs, 263 cubic millimetres of fluid were ejected into the scale-pan. On raising the trunk by the legs 255 cubic millimetres were ejected. When the body was bent far backwards 1,500 cubic millimetres ejected. When the body was bent far forwards 258 cubic millimetres ejected. When the body was rotated by the shoulders either to left or right 78 cubic millimetres ejected.

II. *October 30, 1889.*—Cadaver of male child, æt. seven years; cause of death diphtheria. Trephined at vertex to right of median line.

Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 450 cubic millimetres; bending the body backwards caused ejection of 530 cubic millimetres; bending the body forwards caused ejection of 215 cubic millimetres: rotation by the shoulders to right or left caused ejection of 180 cubic millimetres.

Body hanging as before but with the cervical spine fixed against bar:—Bending the body backwards caused ejection of 215 cubic millimetres; bending the body forwards caused ejection of 180 cubic millimetres.

III. *February 14, 1890.*—Cadaver of woman, æt. twenty-

eight years; cause of death, pericarditis. Trephined at vertex to right of median line. Weight of cadaver, 7 st. 4 lbs.

Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 98 cubic millimetres; bending backwards the body caused the ejection of 480 cubic millimetres; bending the body forwards caused ejection of 220 cubic millimetres; rotation by the shoulders right or left caused ejection of 110 cubic millimetres.

Body hanging as before but with cervical spine fixed against the bar:—Bending the body backwards caused ejection of 230 cubic millimetres; bending the body forward caused ejection of 200 cubic millimetres; bending the trunk backwards through an angle of  $40^\circ$  against a fulcrum two inches below the inferior angle of the scapula caused an ejection of 2,050 cubic millimetres; bending the trunk forwards through an angle of  $45^\circ$  over the fulcrum at same level as last gave a displacement of 20 cubic millimetres.

IV. *November 20, 1889.*—Cadaver, male, weighing 11st. 4lbs., æt. thirty years; cause of death, typhoid.

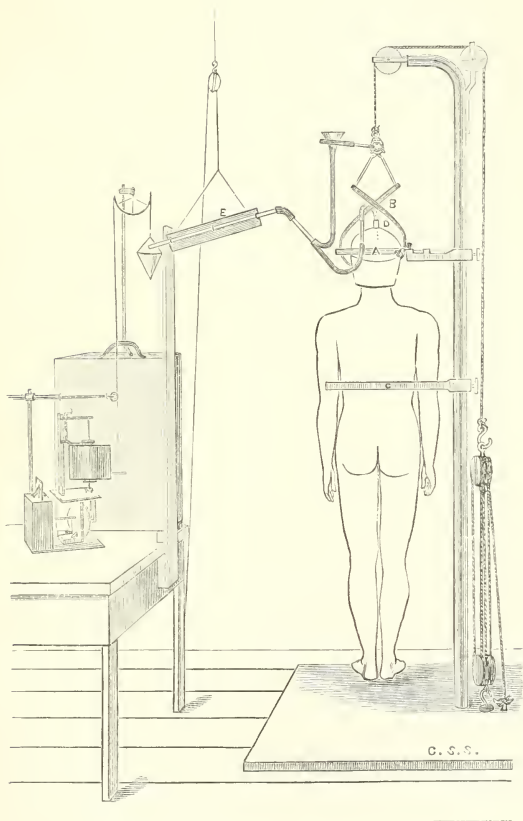
Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 190 cubic millimetres; bending the body backwards caused the ejection of 1,025 cubic millimetres; bending the body forwards caused the ejection of 600 cubic millimetres; rotation by the shoulders to right or left caused ejection of 100 cubic millimetres.

Body hanging as before, but with cervical region of spine fixed against the bar:—Bending the body backwards gave ejection of 450 cubic millimetres; bending the body forwards gave ejection of 150 cubic millimetres; bending trunk backward through  $40^\circ$  over a fulcrum two inches below inferior angle of scapula gave ejection of 2,400 cubic millimetres; bending trunk forward through  $45^\circ$  over fulcrum at similar level gave ejection of 28 cubic millimetres.

V. *November 16, 1889.*—Cadaver, male, powerful frame, æt. thirty-six years, weight 11st. 2lbs.; cause of death, pneumonia.

Body hanging freely from the head:—Raising the trunk by the pelvis caused the ejection of 70 cubic millimetres;

TO ILLUSTRATE PAPER ON THE EFFECT OF  
MOVEMENTS OF THE HUMAN BODY ON  
THE SIZE OF THE SPINAL CANAL.







bending the body backwards caused the ejection of 1,800 cubic millimetres; bending the body forwards caused the ejection of 920 cubic millimetres; rotating the shoulders to right or left caused ejection of 100 cubic millimetres.

Body hanging as before, but with the cervical spine fixed against the bar:—Bending the body backwards caused ejection of 800 cubic millimetres; bending the body forwards caused ejection of 260 cubic millimetres; bending trunk backwards through  $40^{\circ}$  over fulcrum two inches below inferior angles of scapula gave ejection of 1,250 cubic millimetres; bending forward through  $45^{\circ}$  over fulcrum at same height gave ejection of 30 cubic millimetres.

VI. *November 15, 1889.*—Cadaver, male, weighing 12st. 4 lbs. Cause of death, cardiac disease.

Body hanging freely from the head:—Raising the trunk by the pelvis caused ejection of 48 cubic millimetres; bending the body backwards caused ejection of 1,200 cubic millimetres; bending the body forwards caused ejection of 250 cubic millimetres; rotation by shoulders to right or left caused ejection of 140 cubic millimetres.

Body hanging as before, but with cervical spine fixed against the bar:—Bending the body backwards gave ejection of 900 cubic millimetres; bending the body forwards gave ejection of 200 cubic millimetres; bending body backwards through  $40^{\circ}$  over fulcrum two inches below inferior angle of scapula, gave ejection of 2,300 cubic millimetres; bending body forward through  $45^{\circ}$  over fulcrum at similar level gave ejection of 36 cubic millimetres.

VII. *October 22, 1889.*—Cadaver, female; weight, 7 stone 10 lbs.; æt. fifty-four years. Trauma.

Body hanging freely from the head:—Raising the trunk gave ejection of 90 cubic millimetres bending the body backwards caused ejection of 280 cubic millimetres; bending the body forwards caused ejection of 145 cubic millimetres; rotating the shoulders to right or left gave ejection of 95 cubic millimetres.

Body hanging as before but with cervical region fixed against bar:—Bending body backward gave ejection of 170 cubic millimetres; bending body forward gave ejection of

40 cubic millimetres; bending trunk backward through 40° over fulcrum at inferior angle of scapula gave ejection of 1,100 cubic millimetres; bending forwards through 45° over similar fulcrum gave ejection of 32 cubic millimetres.

QUANTITY OF FLUID DISPLACED FROM CRANIO-VERTEBRAL CANAL DURING MOVEMENTS OF THE BODY.

I.—CADAVER HANGING FREELY FROM THE HEAD.

Age in Years.	Sex.	Relieving the spine from the weight of the body by lifting.	Bending Body.		Rotation by the shoulders.
			Backward.	Forward.	
4	♂	Cub. Mil. 263	Cub. Mil. 1500	Cub. Mil. 258	Cub. Mil. 78
7	♂	450	530	215	180
28	♀	98	480	220	110
30	♂	190	1,025	600	100
36	♂	70	1,800	920	100
45	♂	48	1,200	250	140
54	♀	96	280	145	95

II.—CADAVER SUSPENDED FROM HEAD, BUT WITH CERVICAL REGION OF THE SPINE PREVENTED FROM BENDING BY A BAR.

Age in Years.	Sex.	Bending Backward.	Bending Forward.	Bending of spinal column over a bar at level close below the inferior angle of the scapula.	
				Backward.	Forward.
7	♂	Cub. Mil. 215	Cub. Mil. 180	Cub. Mil. —	Cub. Mil. —
28	♀	230	200	2,050	20
30	♂	450	150	2,400	28
36	♂	800	260	1,250	30
45	♂	900	200	2,300	36
54	♀	170	40	1,100	32

Conclusions that we arrive at from our observations may be shortly set forth as follows :—

1. That when the body hangs freely and vertically from the skull the capacity of the cranio-vertebral canal is at a maximum.

2. That with the body in the above position, when the weight of the trunk and limbs is taken off by, for instance, lifting and supporting the body vertically, there is a diminution in the capacity of the cranio-vertebral canal, but the diminution is a very slight one.

3. That when the vertebral column is bent backward or forward, especially in the former direction, there is a not inconsiderable diminution in the capacity of the cranio-vertebral canal as compared with its capacity when the body is hanging freely and vertically.

4. That the alterations in the curvatures of the spinal canal by various movements of the body do influence the capacity of that canal, but not to any great extent; much more however in the child than in the adult.

A point of practical interest may be adverted to. From the above measurements it would appear that by suspension the size of the spinal canal is increased in the adult of middle age to the extent of some 100 cubic millimetres. Estimating the total capacity of the canal at 102 ccm., this measurement means an increase in the size of the canal to the extent of 1-1020·0th of the whole canal. This increase is so small that it becomes difficult to conceive how, in the “suspension” treatment lately introduced for cases of *tubes dorsalis*, &c., there can, as has been sometimes claimed, be any actual stretching of the spinal cord.

## Clinical Cases.

### NOTES ON THREE CASES OF AN HEREDITARY FORM OF PROGRESSIVE AMYOTROPHY.

BY H. B. DONKIN, M.B. OXON, F.R.C.P.

*Physician to Westminster Hospital, and to East London Hospital for  
Children.*

THE cases described below were shown by me at the Neurological Society, in June, 1890, and were considered by most observers to be fitly classed with those described by Leyden in 1875, in which the symptoms are noted as beginning in the lower extremities. In 1884 Dr. Ormerod<sup>1</sup> published three very similar cases in one family under the heading of "cases of muscular atrophy after measles." In 1886 MM. Charcot and Marie<sup>2</sup> described an affection of this kind; in the same year Dr. Tooth<sup>3</sup> dwelt on the subject at length, with cases, under the title of the "Peroneal Type of Muscular Atrophy." His papers are the English *locus classicus* of this matter up to the present time. Dr. Herringham<sup>4</sup> gave an important and detailed account of one case in 1888.

CASE I.—Walter Rivitt, aged nine years, admitted into the East London Hospital for Children in June, 1890, for difficulty in walking. He is a healthy-looking boy, and there is no evidence of any disease other than is mentioned below. (The fundi oculorum are normal.) His gait is high-stepping, not spastic; the cause being foot-drop owing to weakness of muscles of dorsal flexion, so that the leg has to be raised high for the toes to clear the ground. The feet are in the position of partial varus, both at rest and in walking. This is most marked on the left side. The absence of any contraction shows that it is due to peroneal weakness. In walking the ball of the foot is brought down suddenly on the ground before the heels. All the muscles of the legs below the knees are evidently wasted, while the thigh muscles are appa-

<sup>1</sup> BRAIN, Oct., 1884.

<sup>2</sup> *Revue de Médecine*, Feb., 1886.

<sup>3</sup> BRAIN, vol. x., p. 243, and *St. Bartholomew's Hospital Reports*, vol. xxv.

<sup>4</sup> BRAIN, July, 1888.

rently normal. Sensibility to pain, as tested by pricking, seems to be decidedly diminished over the leg-surface, and a faradic current causing great pain elsewhere is here said to be unfelt. Similar insensibility is apparent even after a prolonged application of the negative electrode of a galvanic battery. There is no reaction to faradism or galvanism in any of the muscles below the knees, while the thigh-muscles react quite normally. The knee-jerks are present, and there is no ankle-clonus.

The upper extremities show the ordinary marks of progressive muscular atrophy affecting the hands; there is a moderate degree of the *main-en-griffe*, and considerable wasting of the thenar and hypo-thenar muscles. There is the same apparent loss of sensibility over the skin of the hands, and there is no reaction to faradism or galvanism in any of the short muscles of the hands, except perhaps very slightly in the first and fourth dorsal inter-ossei. The forearm muscles respond less readily to faradism on the extensor than on the flexor aspect. There is no reaction of degeneration anywhere, and the muscles of the rest of the body react normally in every respect.

This boy's *personal history* was that the weakness in the ankles began two years before admission, very soon after an attack of measles, and has lately become rapidly worse. The hands were first noticed to be weak about six months ago. He had measles twice, the weakness following on the second attack; chicken pox and inflammation of the lungs when very young; no fits or other illness. The mother states that he has always been intelligent and very healthy. He seems perfectly intelligent now.

The *family history* is that the father, a carman, is healthy, but walks like the patient, and has "contracted hands." The mother is healthy. She had eight children; two were still-born, two died of "fits" in infancy, and of the four living a sister, aged seventeen, suffers like her brother and father. The mother had two miscarriages after the patient's birth. There is no history or evidence of syphilis in any of the family. The patient's paternal great-grandfather "suffered in his hands" in the same way. No further history of nervous or other disease.

CASE II.—Alice Rivitt, the above-mentioned sister, aged seventeen years, came to the hospital at my request, and was examined by Dr. Hastings, the resident medical officer. The gait and wasting are almost exactly the same as in her brother's case, but the wasting is more marked in the upper extremities. The wasting is said to have begun in the legs at the age of seven (as in the brother's case) and also just after a second

attack of measles. The wasting of the hands was observed two years later. For several years now she has not been getting worse in any respect.

The knee-jerks are present, and there is no ankle-clonus; there is loss of sensibility of the same distribution as in the brother's case. The muscles below the knees fail to react; the thigh muscles react, but less readily than the brother's. In the right forearm the long extensor of thumb and both carpal extensors react, but no short muscles. In the left forearm all extensors of thumb and wrist react, but imperfectly; the extensors of fingers do not react. All flexors of forearm except those of thumb react, but none of the short muscles. The muscles of both upper arms react somewhat imperfectly, but all others are normal. No fibrillar or other tremors have been observed in either of the patients.

CASE III.—The father of the above two patients, æt. forty-two, was also examined electrically by Dr. Hastings. He stated that he was about seventeen when the weakness appeared in his legs, and that the hands began to suffer in the same manner as those of his children about two years afterwards. He does not remember any previous illness. For many years he has been as bad as he is now, with marked deformity of feet and hands. On the left side the leg below the knee is very much wasted, all the muscles being affected, but especially the anterior tibial and the peronei. The foot is in the equinus or slightly equino-varus position, and is much hollowed out on the inner side. There is no power of flexing the ankle, but he can extend it after passive flexion. There is no movement of the toes. The right leg is similar, but the wasting is everywhere less, and the muscles have more tone; the movements, however, are but very slightly better. Both upper arms are well nourished, and their movement normal.

To faradism the extensors of right forearm react, but the *E. carpi ulnaris* less so than the rest; both flexors of the wrist act, but none of the short muscles of hand to either current. On the left side all the forearm muscles act normally, but none of the hand muscles. Below the knees there is no reaction to either current, and the patient does not feel any pain on the application of the electrodes. The left trapezius muscle shows a slight degree of R.D. The other muscles are normal. The knee-jerks on both sides are very brisk, and there is no ankle clonus. No tremors anywhere, and no marked loss of sensibility to pricking. The man underwent tenotomy for his feet at the age of twenty as a surgical patient at one of the London hospitals.

*Remarks.*—These cases show the closest resemblance to Dr. Ormerod's, which were also of a father and two children. All six began in early life, the electrical reactions were practically the same in all, and there is a history of measles immediately preceding the affection in five. The father in Dr. Ormerod's cases was, however, affected in one leg only, and from this fact and the want of opportunity of complete examination, Dr. Ormerod hesitated to pronounce a definite diagnosis in this instance.

In Dr. Herringham's case there were fibrillar tremors, no apparent alteration of sensibility, and only diminished reaction to faradism, with no R.D.; but many features of the case seem to be the same. Dr. Herringham traced the disease through five generations, the transmission appearing to take place through females to males alone.

Dr. Tooth's collection of cases shows that heredity, though frequent, is not always present. He also mentions marked pains in some instances, and tremors, with faradic contractility and unaltered galvanic reactions in the early stage.

It must be remembered that Dr. Ormerod's and my own cases, where there was no reaction to either current in the affected muscles, were mostly of many years' standing; yet two of them, boys of nine years old, showed this characteristic after the disease had lasted two and four years respectively.

It seems probable that the occurrence of measles played the part of an immediately exciting cause, or occasion, of the neuro-muscular breakdown which marks these cases. There appears to be no definite characteristic by which either a myelopathy or a peripheral neuropathy or a myopathy can be established or excluded as the original morbid process. The absence of fibrillar tremors probably points *per se* to a myopathy, but there is a want of observation on this point in the early period of the disease. The marked absence of reaction to both currents, which is only known with certainty as obtaining in the advanced stages of the disease, does not give much aid in localizing the original lesion. Whether all the cases I have referred to are to be regarded as coming under one general category can only be settled after more knowledge has been gained from clinical observation of the affections at their outset. Further research on this point, and perhaps fresh facts supplied by morbid anatomy, may possibly in time throw some light on the vexed question of the neuropathic or myopathic origin of any, or all, of such cases—if, indeed, that question be a soluble or scientific one at all. In ultimate ætiological analysis it is

probably a vain and merely scholastic speculation to inquire as to the exact point where neuropathy and myopathy meet or depart, clear though the difference may be between centrally and peripherally-caused neuro-muscular affections as a whole.

The above cases bear some general clinical likeness to pseudo-hypertrophic paralysis, and their exact pathology is about equally obscure.



## A CASE OF RECOVERY FROM CEREBRO-SPINAL MENINGITIS.

BY W. HALE WHITE, M.D.

*Physician to Guy's Hospital.*

JAMES CLEMENTS, æt. 19, living at 15, Earls Road, Old Kent Road, a railway servant, was admitted into John Ward, Guy's Hospital, 29th June, 1889, complaining of severe pain in the back of the head. His mother has paralysis agitans, and his sister is said to have fits.

He first felt ill 27th June, 1889, when he became giddy. He has vomited several times since, and has complained of headache. There is no history of any disease of the ear or nose, nor of any blow.

*On Admission.*—The head is kept rigidly fixed in the middle line, and is slightly retracted. There is much headache and considerable pain and tenderness over the upper cervical spines. He does not move his trapezius when he attempts to shrug his shoulders. Pulse varying in rapidity, rarely more than fifty-eight, often a little less, irregular, occasionally it intermits. The pulse and temperature have lost their mutual relation, thus with the pulse at fifty-eight the temperature was 102° F. Respiration irregular, twenty-two to the minute. Sometimes the diaphragm alone is in action, at other times only the costal muscles, the two rarely acting together. Breath sounds normal.

The tongue is coated with a slight fur. There is little appetite, the bowels have not been open since June 26th. The urine is healthy.

*Nervous System.*—The patient is delirious at times; he complains of great headache. He says the limbs are weak, but the grasp is fairly good. There is no obvious paralysis in any part of his body, but certain movements of the head cause pain. There is no anæsthesia nor hyperæsthesia. The lower edge of the right optic disc is slightly blurred, and the whole surface is redder than normal. The left optic disc is much blurred and red. There is

photophobia. The pupils are equal and react normally. The superficial and deep reflexes are all normal.

Ice bags were applied to the head and neck. A single dose of a minim of croton oil was given. The patient was put upon moderate doses of Hydrargyrum cum Creta, which was continued during the whole of his stay in the hospital. Milk diet was ordered.

*June 30th.*—Patches of herpes have appeared round the mouth on the left side only; there are no signs in the lungs to account for this. All the symptoms remain the same, except that the fixation of the neck and the pain are not quite so severe.

*July 3rd.*—The symptoms are the same, except that in every respect the patient seems much better.

*July 4th.*—The patient is worse again. He is delirious, he mutters continually, and he allows flies to collect on his face. The breathing is very irregular. He passes his urine and fæces under him. The optic discs remain the same as on admission. It is difficult to tell whether he has much pain. The pulse is slow and irregular. He vomits a good deal, and the vomiting is characteristically cerebral. Although the patient is so much worse, the temperature is only 98·8 F.

*July 5th.*—Is in much the same condition as yesterday.

*July 6th.*—Is worse in every respect, save that the temperature remains normal. The symptoms are unchanged, but there is in addition decided external strabismus in the left eye.

*July 10th.*—Since the last note the patient has been improving in every respect. He is now rarely delirious; the headache is better. He is very weak, but takes milk well. The temperature is about normal.

*July 12th.*—This morning there is a change for the worse. The patient is unconscious, he has muttering delirium, and passes everything under him. He takes no notice of anybody nor anything. He still has strabismus. His pulse is irregular and slow.

*July 17th.*—During the last five days the patient has been so ill that it has always seemed almost impossible that he can survive more than a few hours. The symptoms have remained the same as since the last note, except that they are much worse, and he has been much troubled with hiccough.

*July 19th.*—For the last two days the patient has been much better. He has become conscious, and no longer passes his urine and motions under him. He is as thin and wasted as any patient I have ever seen. From this date he gradually improved, very slowly at first but more rapidly afterwards, so that by August

20th he was gaining 2lbs. a day. All his symptoms disappeared before he left the hospital at the end of September, except the strabismus.

*Nov. 21st, 1889.*—The patient was seen to-day. He says he can walk any distance, and he has gone back to his work as a truck shunter on the railway. Since he left the Hospital his weight has increased from six and a half stone to ten stone. The only thing he complains of is that he occasionally gets a pain down the back, but this trouble is not severe. He has no headache. The optic discs are perhaps a trifle red, but they are hardly abnormal. The squint that he had when he left the Hospital has disappeared, but the movements of the eyes are not always perfectly free. The pulse is regular. It is interesting to note that he has a slight patch of herpes on the lip in the same place as the herpes broke out when he was in the hospital. His knee reflexes can only be obtained with difficulty. He is quite sure he never had herpes before this illness.

*January 17th, 1890.*—Re-admitted into Guy's Hospital under Dr. Taylor. About a month ago he began to notice that his right leg was getting weak; this has gradually become worse. His right arm has been getting weak for the last ten days. Since his discharge he has never suffered from headache or sickness, nor has he had any fits.

*Condition on admission.*—Heart, lungs, urine, and temperature all normal. He looks healthy. He is very emotional, weeping about the loss of power in his leg. Intelligence and memory good.

*Sensation.*—There seem to be slight impairment and delay of sensation all over right limbs and the right side of his body. There is no analgesia, nor paræsthesia. Hearing, sight, and optic discs normal. Muscular sense for arms, the same on the two sides. Differences of 1 in 13 are rightly distinguished.

*Motion.*—Superficial reflexes normal, no knee jerks, no ankle clonus. On smiling, the right side of the face moves slightly less than the left, and the tongue is protruded slightly to the right. Movements of respiration normal. Loss of power in both arms, especially the right. With the dynamometer the right grasps 50, the left grasps 60. No difference in measurements, no rigidity. Loss of power in both legs, especially the right.

Right thigh, 16 inches in circumference.

Left „ 15½ „ „

Right calf, 10¾ „ „

Left „ 12 „ „

Left gluteus maximus seems smaller than right.

The loss of power is most marked in both anterior tibial muscles, and in the right rectus. His gait is very unsteady, but it is not characteristic of any particular form of disease. The electrical reaction of all the muscles are normal, except that in the flexors of the right forearm, ACC>KCC.

He went out on February 18th, 1890, having in all respects rather deteriorated since admission.

During the summer of 1890 he was for some time in the hospital at Queen's Square.

Early in October, 1890, I saw him. He was still weak in his legs, and his gait was staggering. He was also lachrymose.

*November 20th*, 1890.—I saw the patient to-day, he is now much better. There is no ocular paralysis; both the legs and arms are rather weak, but he can walk a long distance, although his gait is still a little unsteady. His knee jerks are normal. He does not complain of any sensory symptoms, but he is inclined to cry. On the whole he is much better.

## PARALYSIS AGITANS IN A YOUNG MAN.

BY W. B. HADDEN, M.D., F.R.C.P.

THE patient was a man, aged twenty-eight, a shoemaker by trade, who was sent to me by Mr. A. L. Perkins, of Sketty, South Wales.

There was nothing of importance to note in the family history.

When two years old he had an attack of infantile paralysis, and when fifteen years of age he had typhoid fever. When he was twenty-five he had an attack of what, from his description, would appear to have been acute rheumatism. Three months later the left hand began to tremble, and since the onset the movements have gradually become worse.

The patient was a healthy-looking man, with a fixed expression of face. There was no marked defect of articulation, but when he spoke there was little movement of the face beyond the immediate neighbourhood of the mouth. This was made especially evident by covering up the mouth and chin, and then getting him to speak. The body and head were not inclined forward, as is often the case in paralysis agitans. There were no tremors of the head or tongue, and no nystagmus. When directed to look to one or other side the movements of the head and eyeballs were synchronous. The left upper extremity was slightly flexed at the elbow, and the forearm in a position midway between pronation and supination. The whole limb was affected by rhythmical movements of abduction and adduction. The metacarpo-phalangeal and phalangeal joints were slightly flexed, and the digits were the seat of constant to-and-fro movements, which were much increased when he was excited, or when he was being watched. There was alternate flexion and extension of the wrist simultaneous with the movements of the fingers. When on the point of taking a glass of water the movements diminished for the moment, but increased again when the glass was seized, and became very marked as it was carried to the mouth. The movements ceased entirely during sleep. With the

left hand he was slow in performing voluntary movements, such as buttoning and unbuttoning, and in threading a needle (he was a shoemaker). There was no coarse muscular weakness, and no loss of sensation of the affected limb. There were no movements elsewhere in the body. The right lower limb was considerably wasted, though no special muscular groups were picked out. There was double pes valgus; the knee-jerks were feeble. When walking he waddled from side to side, and limped with the right leg, but his gait was not slow, and there was no tendency to festination.

*Remarks.*—I have no doubt the case was one of true paralysis agitans. I had intended showing the patient at the Neurological Society, but when he found that treatment was without effect he declined to stay longer in the hospital. My friend, Dr. Beevor, saw the man, and confirmed the view which I had taken. The onset of paralysis agitans at the age of twenty-five is very exceptional, but a few cases have been recorded even at an earlier age.

## Critical Digests.

### FRIEDREICH'S DISEASE.

BY DR. P. LADAME.

(Read before the Medical Society of Geneva. Translated by W. Dudley, M.B.)

DUCHENNE de Boulogne\* had just published his classical study on progressive locomotor ataxy (1858 and 1859). This work aroused great interest, and immediately gave rise to numerous controversies, of which we still hear the last echoes. Duchenne has often been reproached, and with reason, with having caused an unfortunate confusion by this name, "locomotor ataxy," from his taking the symptom for the disease. Jaccoud† one of the first, in a historical critique too severe to be fair, accuses Duchenne of having fallen into an error, carefully avoided in recent times, when he named this disease from the functional trouble, from the symptom which it causes. "It is," says Jaccoud, "a nosological error of the same kind as if one called pulmonary phthisis, chronic cough; pneumonia, acute cough; or myelitis, paraplegia."

Nothing discloses this error better than the history of the disease described for the first time by Friedreich, September 18th, 1861, at the Congress of German naturalists and physicians at Spire. The eminent clinician of Heidelberg had indeed observed with the greatest care several cases of locomotor ataxy, the symptoms of which differed greatly from those which had been described two years before by Duchenne in the *Archives générales de Médecine*. Now Friedreich, who frequently had the opportunity, in

\* DUCHENNE DE BOULOGNE, De l'ataxie locomotrice progressive. *Arch. gén. de méd.*, Dec. 1858, Jan., Feb., April, 1859.

† S. JACCOUD, Les Paraplégies et l'Ataxie du mouvement. Paris, 1864, p. 383

1858 and 1859, of showing his cases to several of his colleagues, among others to Eisenmann, Hasse, Kussmaul and Virchow, accepts only with reserve the clinical picture of Duchenne. He protests even against the pathological type created by the latter. "The complexus of symptoms," says he, "which constitutes progressive locomotor ataxy of Duchenne, cannot absolutely be considered as representing a distinct disease *sui generis*. It is a morbid entity, a group of symptoms which has its anatomical and physiological characteristics, but it is not a special disease, for progressive locomotor ataxy, the failure of co-ordination of voluntary movements, presents very great variations." Witness the cases observed and described by Friedreich, which offer very remarkable differences from those of Duchenne.

Friedreich, with his clinical insight, had therefore correctly guessed that his cases did not come under the description of locomotor ataxy of Duchenne. He did not think, however, that he ought to separate them entirely. He thought that the picture of Duchenne was incomplete, and as one of the fundamental differences between his cases and those of the great French pathologist was found to consist in the absence of sensory disturbances, he adopted the following hypothesis, which might, according to him, furnish a satisfactory explanation of it.

"If, therefore," says Friedreich (<sup>3</sup>), "in the different cases of locomotor ataxy which have been published, we find sometimes sensory disturbances, sometimes complete absence of them, it is because, in the first case, degeneration of the medullary fibres is not limited to the posterior columns, but has invaded other regions of the spinal cord."\*

When he put forward this hypothesis, ingenious rather than well founded, Friedreich had already made three autopsies, the results of which would have permitted him to conclude that there might be combined sclerosis of the posterior and lateral columns, since this lesion manifestly existed in two of his cases.

Nevertheless, many years will pass before the ataxy of

\* JACCOUD, *loc. cit.* p. 677, adopts this erroneous hypothesis.



Friedreich takes rank in nosography. For a long time, quite recently even, we have seen it, this morbid form will be considered by the authors, Topinard, Erb, Eulenburg, Jaccoud, Möbius, Grasset, Strümpell, &c., as a variety of *tabes dorsalis* (locomotor ataxy of Duchenne). To-day even, it is in the chapter on this affection that we must look for the description of *hereditary ataxy* in pathological works.

¶ Hereditary ataxy! That is, indeed the name chosen by Friedreich as the title of his second memoir. The disease is not observed, he thought, in isolated individuals. Each time that he had seen a case of it he met with several others in the same family amongst the brothers and sisters of the patient. Eisenmann,† who speaks nowhere of Friedreich's cases, was the first to employ the words hereditary ataxy (*erbliche ataxie*), quoting as an example the case observed by Carre, and the opinion of Trousseau. This name of hereditary ataxy has been justly criticised. M. Féré<sup>(33)</sup>, following Dr. Gowers<sup>(25)</sup>, of London, and Brousse<sup>(34)</sup> of Montpellier, has shown that it was "manifestly wrong," since, on the one hand, classical locomotor ataxy recognised also very frequently for primary cause, either direct heredity, or more often neuropathic heredity, and, on the other hand, Friedreich's disease is absolutely distinct from locomotor ataxy, and consequently should not be confounded with it under a common name. Moreover, it is not heredity which is the essential and striking feature in this affection; it is rather its "familial" character, that is to say, the frequency of the development of the disease in several children of the same family. Dr. Everett Smith<sup>(60)</sup>, of Boston, proposed for this reason to call it "postero-lateral spinal sclerosis of generic origin," or more simply "generic ataxy." This name, however, does not suit a disease which has nothing in common with progressive locomotor ataxy, as Pitt<sup>(74)</sup> says, except the abolition of the knee jerks.

After having been regarded as a particular form of locomotor ataxy of Duchenne, Friedreich's disease was also considered by some authors as a simple variety of dissemi-

† EISENMANN in Würzburg, *Die Bewegungs-Ataxie*. Wien, 1863, p. 205.

ated sclerosis, or at least as a combination of locomotor ataxy and multiple sclerosis. Charcot\* and Vulpian† had in fact just described the symptoms of disseminated sclerosis. Everywhere interest was taken in the strange manifestations of this affection, of which a learned diagnosis was gladly made in medical clinics.

An attentive study of Friedreich's cases made M. Charcot at first think that perhaps he was dealing with disseminated sclerosis having invaded principally the posterior columns.‡ Such a supposition must indeed have presented itself naturally to the mind of observers of that period, who had never seen cases analogous to those of Friedreich. Hence, M. Bourneville examines in a special chapter§ Friedreich's cases under the title, "Cases in which there exist simultaneously the symptoms and the lesions of locomotor ataxy and of disseminated sclerosis."

After having given the translation of two of the principal cases of Friedreich, M. Bourneville expresses himself as follows:—

"The mode of onset indicated is not that which is ordinarily seen in ataxy, whilst it is much more conformable to that of disseminated sclerosis. Difficulty of speech, trembling of the head, nystagmus, are morbid phenomena foreign to the classical type of ataxy. Finally, contrary to what takes place so frequently in this disease, cutaneous sensibility and vision were not affected."

And with regard to the second case:—"The onset here has occurred in the same way as in the first case. . . . Moreover, we see that the patient has had spasmodic contractions in the peronei muscles, stammering, nystagmus, enfeeblement of motor power and of vision, and moreover that cutaneous sensibility was intact.

\* CHARCOT, *Leçons sur les maladies du système nerveux*, t. I (Clinical lecture of 1868). See Bourneville's digest in *Mouvement médical*, 1868, Nos. 197, 235, 259 et 327.

† VULPIAN, *Note sur la sclérose en plaques de la moelle épinière*. *Union médicale*, 1866.

‡ See FETZER'S "Klinische Vorträge von Charcot," Stuttgart, 1874, p. 521 quoted by Friedreich.

§ BOURNEVILLE, *Nouvelle étude sur quelques points de la sclérose en plaques disséminées* in BOURNEVILLE et L. GUÉRARD, *De la sclérose en plaques*, p. 191 Paris, 1869, chap. ii., p. 204.

“On the other hand, apart from lightning pains, there is nothing clearly belonging to ataxy, for the motor disturbances are so badly defined that one might ask if we are concerned with the trembling of disseminated sclerosis or with the inco-ordination of locomotor ataxy. . . .”

And M. Bourneville concludes:—“What is the outcome of this? It is that incontestably there is a mixture of two diseases . . . to wit, the co-existence in this case of progressive locomotor ataxy and of disseminated sclerosis.”

Professor Charcot does not admit this combination of the elementary forms of these two diseases. “For myself,” says he, in his *Lectures on the Diseases of the Nervous System*, “I have never met with, on the cadaver, the co-existence of multiple grey induration and posterior *fasciculated* sclerosis, and without denying that this association might exist, I believe it to be at least infinitely rare. It is pretty common, on the contrary, that the sclerous patches, which, as a rule, are principally seated in the antero-lateral columns, pass across the postero-lateral fissures, and encroach upon the posterior columns. Sometimes even I have seen them, having become continuous, occupy a large part of the thickness of these columns in the whole extent of one of the regions of the spinal cord—the lumbar, for example. Now, in all the cases of the latter kind, the ataxic symptoms had been, during life, manifested in different degrees.”\* And M. Bourneville adds in a note that the two cases of Friedreich, referred to above, belong to this category, what Charcot had not said expressly.

Friedreich<sup>(1)</sup> refutes this opinion by close reasoning, and demonstrates victoriously that his cases do not belong to disseminated sclerosis. “Why,” says he, “has Bourneville made choice in my cases, reproducing some, and leaving others out? It is very surprising, for example, that he does not mention my first patient, Andreas Lotsch, since in this case the clinical and anatomical characters of degeneration of the posterior columns were certainly of the clearest.” Friedreich then asks himself, by what signs one ought to

\* CHARCOT, *Leçons sur les maladies du Système nerveux*, t. I, seconde édition. Paris, 1875, p. 243.

recognise "a true ataxic." Should it be, as Duchenne has laid down, by the absence of speech troubles? But one might upset this statement by arguing, on the other hand, that *true ataxics* are those in whom the want of co-ordination extends to speech and to ocular movements! "In my opinion," says Friedreich, "Charcot has attributed in my cases too great importance to nystagmus and to disorders of speech, whilst all the other more characteristic signs of disseminated sclerosis have been absolutely wanting during the many years that the patients have been under my observation. They have never presented diplopia, amblyopia, muscular contraction, spinal epilepsy, epileptic or apoplectic attacks, intellectual disturbance, bedsores, &c. The ataxic nature of the motor disturbances of my patients was so characteristic that it was impossible to doubt their significance, and because the ataxy invades the muscles of the tongue and those of the eyes, would that be a reason for not considering them true ataxics! Is it not, on the contrary, still more a reason for considering them as such?"

"I find it incomprehensible," says Friedreich finally, "that anyone can still speak of disseminated sclerosis, when I have given the results of three carefully-made autopsies . . . and I hope that Charcot, in the vast field of observation which he commands, will sooner or later find a case analogous to those I have described."

This prediction was to be realised eight years later. I shall remark, moreover, that Charcot had never himself definitely pronounced on Friedreich's cases. He remained rather expectant, as we see by the quotations borrowed from his lectures. But in 1884, in one of his clinical lectures at the Salpêtrière, he comes boldly from his scepticism, and shows a young patient affected with hereditary ataxy, which he for the first time considers as a special affection, clearly differentiated from disseminated sclerosis and from locomotor ataxy. One may say that from that moment Friedreich's disease was clinically recognised in official teaching; henceforth it will occupy a distinct place in nosography among spinal affections.

"This disease," says Charcot, "approaches in symptoms

at the same time, locomotor ataxy properly called and disseminated sclerosis, whilst it distinguishes itself from them entirely in certain directions; it has been called hereditary ataxy, or Friedreich's disease" (45).

Two years before had appeared at Montpellier the important work of Dr. Auguste Brousse, who was the first to propose the name of "Friedreich's disease," to designate the particular morbid affection so well described by the professor of Heidelberg. This work gives a complete *résumé* of the subject at that time. It appears interesting to reproduce from it the principal conclusions:—

"The disease studied by Friedreich," says Brousse (24), "under the name of hereditary ataxy, is a special disease having a proper etiology, symptomatology, and morbid anatomy. It is characterised—

"1. From the etiological point of view, by its development in childhood or adolescence, due to hereditary influence, direct or indirect; the two sexes appear equally predisposed.

"2. From the symptomatic point of view, by ataxy of the four limbs, commencing in the lower extremities and developing so as to reduce the patient to almost absolute functional impotence; by difficulty of speech, by absence or late appearance of sensory disturbances; by absence of trophic troubles and preservation of tone of the sphincters; by slow and continually advancing progress; by very long duration; finally, by an always fatal termination, generally brought about by an intercurrent disease.

"3. From the pathological point of view, by fasciculated sclerosis of the posterior columns of the cord, extending upwards to the bulb, complicated by diffuse sclerosis of the lateral and anterior columns.

"4. From the diagnostic point of view it must be distinguished from progressive locomotor ataxy, from disseminated sclerosis, and, lastly, from cases described in Germany by Westphal and Schultze under the name of combined degeneration of the columns of the cord.

"5. From the nosographic point of view it renders necessary the creation of a new class of chronic spinal affections—the class of mixed myelitis."

In England hereditary ataxy was first described before the Clinical Society of London, October 8th, 1880. At this meeting Dr. Gowers showed three patients belonging to a family of nine children, five of whom were ataxics. He specially insisted on the characters which absolutely distinguish this disease from ordinary locomotor ataxy. Gowers recalled on this occasion that Dr. Carpenter (<sup>6</sup>) of Croydon, in 1871, had brought before the same Society two sisters affected with a curious disease hitherto unknown, and which was no other than Friedreich's ataxy. Since then, a brother of these two patients has also been attacked by the same disease.

At the meeting of November 13th, 1871, at which Dr. Alfred Carpenter exhibited his patients, the Clinical Society appointed a committee, composed of Drs. Richardson, Hughlings-Jackson, Lockhart Clarke, and Broadbent, to examine these remarkable cases, and to report at the next meeting. These two sisters presented the important symptom of lateral spinal curvature already mentioned by Friedreich, and which has been recognised as one of the characteristic signs of hereditary ataxy.

In recent years, the number of cases of this disease observed in France, Germany, England, Italy and America has so far increased that M. Soca (<sup>91</sup>) in his excellent thesis, in which are recapitulated almost all the known cases, reckons up 165. But that is a very exaggerated total; for if the published cases are more closely examined and seriously criticised, it is soon perceived that the list contains a very great number of cases which are incomplete, doubtful, or even absolutely foreign to Friedreich's disease. Before proceeding to this critical review, which will allow us to eliminate many cases which are inappropriate, and which obscure the clinical picture, it appears necessary for us to fix first of all the essential characters of this picture. To this end I shall give first of all a *résumé* of a case of my own which has been under my care for three years (April 1886), and which is thoroughly typical of Friedreich's disease

A. K., aged twenty-four (in 1886), a member of a liberal profession, has been affected from the age of eight, and has been

unable to walk for the last three years, since he had acute articular rheumatism, convalescence from which was prolonged for several months, at the age of twenty-one.

*Family History.*—Father, aged fifty-six, is very robust, it is said, and has never been ill; I have no information as to his health or his habits. Mother in good health also, but very nervous.

On the paternal side are several uncles and aunts, it appears, who are more or less affected with nervousness and peculiarities of character. There are some “hot-headed” members in the family. But no one is known to have presented symptoms analogous to those of our patient. A younger brother, aged sixteen, only walked when three-and-a-half years old; he is imperfectly developed for his age, very nervous, and of an irritable and troublesome temperament. A younger sister is in good health, but is also nervous.

*Patient's History.*—His early childhood presented nothing particular. Suckled by his mother, the child developed normally, and walked at fifteen months. Very intelligent and precocious for his age, he early learned to read and write. At the age of eight the first difficulty in walking appeared, a certain weakness of the legs and of the movements, which was looked upon as “nervous.” He frequently fell; he walked slowly, and he planted his feet one before the other. He was scolded, because it was looked upon as carelessness and idleness. Nevertheless, he was able to walk easily alone. He never complained of pain. About two years after, he became ungainly with his hands, and five years only after the onset (the first symptoms in the legs) his speech began gradually to become drawling and difficult. The boy was then aged thirteen. Never involuntary grimaces, but so-called nervous movements in the arms and legs during walking. One day when the child was suffering from headache and stomach ache, a medical man was called in, and his visit was taken advantage of to consult him on the singular movements observed in the little patient. The doctor had him undressed, made him walk before him, examined him carefully, and said: “Oh, it will be nothing, it is St. Vitus's dance”—ordinary chorea slightly developed. Now this supposed St. Vitus's dance has never disappeared, on the contrary, it has been accompanied little by little by other symptoms, so that the disease has insensibly become aggravated. For three consecutive years, however, the patient was treated for St. Vitus's dance.

At the age of thirteen, the boy, who was well-advanced at school, was apprenticed to a lithographer, with whom he remained



three years without making any progress. The employer said that he was too weak to follow this trade, and that he would never make anything at it. We must suppose that the ataxy of the arms, which had become more and more developed, was an invincible obstacle to the work of the young apprentice. He was then sent to college, and at the end of some months he had already outstripped his companions by the vigour of his memory and intellect. Henceforth he advanced rapidly and did not cease to devote himself to intellectual labour. However, his gait became more difficult, his handwriting more slow and trembling. At the same time his speech also became slower. At the age of seventeen the patient began to make use of a stick for walking, and henceforth did not give it up. But the progress of the disease had been so insensible and so slow for several years, that he attached scarcely any importance to these symptoms, and the less so as they were not at all painful. He did not perceive, so to speak, that his disease was progressive. Thus he reached the age of twenty-one. At this period he was attacked with acute articular rheumatism, which confined him to bed for three months, and at the end of several months' convalescence he perceived that he could no longer walk alone. In darkness especially he felt less firm on his legs, and for a long time, even before his rheumatic attack, he could not go out in the evening, whilst during the day he still walked pretty well with the assistance of a stick. His legs had become more and more weakened, and even while giving one arm to someone, and helping himself with a stick with the right arm, the patient continued to find more difficulty in walking.

Never has he experienced lightning pains, nor girdle pains. Never incontinence of urine, nor any other urinary trouble. No disturbance of vision. Habitual constipation. No seminal losses. Never syphilitic infection.

*Present Condition.*—(He was examined several times from April, 1886 to April, 1889). Body generally imperfectly developed. Muscular system rather feeble. Adipose tissue scanty. General pallor of integuments. Skeleton fairly formed. Head large, round, symmetrical, brachycephalic. Scoliosis, with convexity to the right very pronounced in the region of the dorsal vertebræ, the ribs bulging on the same side forming a well-marked prominence under the scapula. No other peculiarity to note as to the development of the skeleton except the deformity of the feet which we are about to describe, remarking that it is not of bony nature.

The feet are small, deformed, affected with talipes equinus,



and as if shortened. Their colour is of a darkish blue. They feel cold to the touch. The sole of the foot is much hollowed, and has undergone a certain twisting, which is seen by the transverse furrows of the skin, which have become oblique from within out, and from behind forward, as if the anterior part of the foot had been twisted on the posterior. There is no permanent contraction of the sole of the foot. The dorsum of the foot is prominent; there is a projection at the level of the tarsus. The toes, especially the great toe, are raised up clawlike, and the tendon of the extensor proprius pollicis appears prominently under the skin. These symptoms are especially marked when the foot is raised; they are largely effaced when the patient places his foot on the ground. The articulations are flexible. All passive movements are easily performed. The tibio-tarsal articulation is relaxed, and voluntary extension of the foot can be prevented by fixing it with the hand. During walking the toes are constantly affected with movements of athetosis.

*Motor symptoms.*—The patient cannot stand upright without support. His trunk, head, and limbs are always in motion. He says that he could never be photographed because he is too nervous, and his features are too mobile. In reality it is because he cannot hold still an instant; even when he is at rest, his head and his trunk are incessantly agitated. It is static ataxy, as Friedreich calls it; when seated in an armchair he is perpetually moving. They are a kind of badly marked choreiform movements. We can understand that at the commencement of the affection the disease was mistaken for chorea. We shall speak of it again in treating of the diagnosis. Let us add, however, that this “choreiform instability,” as Charcot calls it, is not always alike. Sometimes very pronounced, it is much less so on certain days, especially if the patient is well rested.

All voluntary movements of the arms and legs can be executed, but are manifestly ataxic. It is impossible for the patient, when told, to touch with his index finger, at the first attempt, the tip of his nose or the lobule of his ear, even with the eyes open. He always places his finger at a certain distance from the point intended, then, without leaving the surface of the skin, he makes it glide slowly to the indicated spot. The patient, whose intelligence is highly developed, is well aware of how his actions proceed. At the moment of raising his index finger to execute the required movement, he knows very well where he is going, but he soon loses control over the direction of his arm, because he has no longer an exact notion of the position of the limb during move-

ment. We see it then perform oscillations which distantly recall the trembling of disseminated sclerosis, but do not sensibly increase in intensity at the final moment. If the patient is told to take up a small object from the table—a pencil, for example, a match, or better still, a pin—he advances his hand above the object on which his eyes are fixed, makes it hover an instant, hesitates, separates somewhat his fingers, then darts suddenly on the object to seize it as a bird of prey would do, with this difference, however, that the direction of the movement is sinuous and interrupted, instead of being direct as in the case of the bird who arrives by a straight line on the coveted object.\*

The method of picking up the pin and the pencil from the table is also characteristic. The patient employs to this end all his fingers, which he keeps separate and then brings together, making the object glide into his hand till he holds it conveniently.

He buttons his clothes with difficulty, especially the small buttons. Nevertheless, even if his eyes are closed, he ends by reaching the small buttons, and when once they are held, he does not relax his hold. He cannot dress or undress without assistance, on account of the effort required to lift his clothes. At the moment of taking off his shirt, when it passes above his head and covers his eyes, he loses his equilibrium completely on the stool on which he is seated, falls backwards and remains with his legs in the air till someone comes to his assistance. However, he has made in the meantime great efforts to regain his equilibrium.

It is the incessant movements of the trunk and limbs which prevent the patient maintaining the upright position. If he is told to loose hold of his support, and he sees around him those who are ready to support him, he can remain standing an instant, his legs widely separated, and even if his eyes are closed he is not more unsteady at first; but his unstable equilibrium is soon disturbed, a more extensive movement of the body makes him lose his balance completely, and he would collapse if not supported immediately (Romberg's symptom).

In spite of this, however, it is easy to assure oneself of the integrity of the muscular sense. The patient distinguishes very readily, with his eyes closed, different coins placed in his hand, in the right hand equally with the left. He estimates also pretty correctly the difference of weights when he supports small objects in the hand.

\* Carre had already observed this movement of the hand, which "hovers" over the object before seizing it, but it is to M. Charcot that we owe the comparison with the bird of prey, which appears to me appropriate and striking.

Dynamometer : right hand forty-one, left thirty.

The patient feeds himself but slowly, and with some difficulty.

The patient writes very well, although rather slowly. The letters are well formed, and not at all shaky. The writing is plain and easily read.

The muscular strength of the arms and legs is well preserved. His leg cannot be flexed with both hands when he maintains it in extension. There is marked enfeeblement of the movements of the feet. Since the patient has been unable to walk alone he thinks he has noticed certain changes in his gait. Formerly he walked more on the soles, the fore part of the foot; now more on the heels, after the manner of ataxics. At present he cannot make a single step without being supported on each side. When he wishes to walk he passes his left arm under the right arm of the person who strongly supports him, and he supports himself with a stick in the right hand. He advances then slowly and with difficulty, making each instant extensive movements of the head and trunk, whilst the inco-ordination of movement of the right arm, which holds the cane, causes the latter to go from side to side so that it often misses the ground, and makes the patient plunge forward at the moment that he felt secure of finding a support.

He drags his feet in walking and controls their movements by sight; at each step the point of the foot deviates inwards, and rubs on the ground, especially the left foot, whilst the heel scarcely touches it. The leg, moreover, is not thrown strongly forward as in ordinary ataxy, for the foot scarcely leaves the ground, and the gait strongly recalls that observed in spastic paralysis, except that the stiffness of the legs does not exist here. The steps are short, unequal and irregular, and when one foot has been put forward the other follows, its fore part being dragged along the ground, deviating strongly into equino-varus, whilst the trunk and the head are thrown back in a disorderly manner. When the foot is raised from the ground the inco-ordination of movement becomes very manifest; the leg is flung forward, and the heel falls heavily. To sum up, what characterises the gait is the fact that the trembling is more pronounced than the ataxy. With the least emotional disturbance, the patient can no longer walk. His legs give way under him. If he is tired his feet continually turn over, and he catches against every stone in the way. He can with difficulty mount stairs, but he descends them with yet greater difficulty.

When I took his foot-prints, after the method of M. Gilles de la Tourette, it was necessary to support him under the arms to

make him walk on the band of paper. This is what explains the absence of the characters which we have just described—the dragging of the front of the foot, and the heel hardly touching the ground. When supported the patient raised his feet, threw forward his legs and brought down the heel at every step. What the print especially verifies is the deep concavity of the sole of the foot, which is shortened, and the trembling, irregular walk of the patient, whose legs double up several times under him during the experiment; lastly, in several prints we find the double heel of ataxics.

The sensibility of the soles was, moreover, so exaggerated that the floor caused an insupportable sensation of cold, and the pressure caused pain; he cried out while taking the first steps with bare feet. The toes were seen to be raised violently in a spasmodic way and the sole to become more hollow each time that the patient raised the foot. The feet very quickly became cold and more and more cyanosed. It was then very difficult to warm them. “When my feet become warm again,” said the patient, “they readily perspire, and quickly lose their heat.” He adds that the impression of cold to the feet was very painful to him and paralysed him. He felt plainly the ground and its inequalities, and did not experience any peculiar sensation in the sole of the feet as is the case in tabetics.

*Disturbances of Speech.*—It was towards the age of thirteen years that difficulty of speech was noticed. His parents remarked that the boy's words became drawling and thick. He did not himself notice it—at least he cannot fix the time when speech troubles first manifested themselves. He does not recall that he may have had an interval between the symptoms in the legs and arms and those of speech. He maintains that they have all been developed almost simultaneously.

His voice is monotonous, very low-pitched. Speech is slow, somewhat scanned, but without hesitancy. The mechanical articulation of the sounds and the duration of the words are alone affected. There is no stammering.

The patient pronounces long words with a single effort, although slowly, and somewhat scanning the syllables; but he has no hesitancy of speech, does not cut the words in two, does not stammer. He will say without stopping, but slowly, i-na-mo-vi-bi-li-té, all in the same drawling tone, no syllables being pronounced more rapidly than others. The small pauses are equal between each syllable. Whilst the general paralytic slurs his syllables, this patient articulates distinctly in a slow, monotonous, and drawling voice.

The tongue, when protruded, is of normal size. But it undergoes very marked ataxic movements; it contracts, it is suddenly drawn to one side, then is withdrawn, curving up behind the dental arch, to come out again with a sudden jerk. His reading is also slow and slurred. The soft palate reflex is absolutely normal. The knee reflexes are completely abolished.

*Nystagmus*.—Horizontal oscillation of the eyes when following a moving object is the last symptom which we should connect with ataxy. Nystagmus is not very pronounced. It must be looked for and brought out to be observed. It is not present when the eyes are at rest. It is a motor ataxy of the ocular muscles, a dynamic nystagmus, in opposition to a static nystagmus, a balancing of the ocular muscles in different directions, which is observed when the eyes are fixed on a point. The patient says that when he fixes an object his sight is soon fatigued. The oscillations of nystagmus are very rapid and of small amplitude; they do not exceed a millimetre in extent, and only take place at the moment when the eyeballs reach the corners of the eyes. However, they are still observed, though more rarely, when the eyes are directed in front; at the moment when the eyes are fixed on the finger placed a few centimetres before the forehead, opposite the root of the nose, some slight oscillations are seen to occur. It is what Friedreich had already clearly observed, remarking that in ordinary cases the existing nystagmus is thus made to cease, instead of being provoked.

*Impulse to laugh*.—The patient is often taken with an impulsive desire to laugh. He notices carefully these occurrences, which come on him unawares, and which he cannot resist. Lips habitually separate.

*Sensibility* absolutely normal in all its manifestations, to contact, pain, temperature and electricity. Nowhere in the legs are anæsthetic patches. He feels everywhere the lightest touch, and localises pin pricks perfectly. Never creeping sensations nor pain in the legs, particularly lightning pains. When the patient walked much, he experienced for several months a pain in the left knee. This articulation has never been swollen. Passive movements have always been free; the knees, left as well as right, supple and flexible. He then experienced great weakness in the legs, following fatigue; he could hardly drag himself along. He felt as if his legs were too long, "the nerves unstrung," especially during the north winds. The pain in the left knee then sensibly increased. He could no longer take long steps as usual. His feet dragged on the ground. This is why the patient

said: "The smaller the steps, the more awkwardly I walk." The pain, which he attributed to a rheumatic origin, has completely disappeared since the patient has been less on his legs, and has scarcely walked at all.

*Pupillary reflexes* perfectly normal to light, to convergence, and to accommodation. No diplopia nor strabismus; sight excellent.

*Sphincters* normal. The patient is not subject to constipation. He can retain his urine for long. No gastric troubles.

*Genital functions*.—No disturbance to note. No involuntary emissions. Erections normal. No impotence nor premature ejaculation.

*Psychical functions* normal. Intelligence remarkable, especially the poetic and imaginative faculties.

*Electric reactions* normal everywhere except in the muscles of the leg (antero-external group), where electro-muscular contractility is weakened.

During electric examination the patient, seated on a stool, cannot be left alone an instant; he loses his equilibrium at every movement, and as he is perpetually in motion he would fall if not watched. (Choreiform instability, static ataxy.) Moreover, he is weighed down, he is doubled up, and his head falls on his breast.

*Galvanic Exploration of Nerves*.—L. ulnar (K.C.C.) 1.5 ma.; R. 1.5. L. radial, 3; R. 3. L. peroneal, 2; R. 3.

*Faradic Exploration of Muscles*.—Tibialis anticus, R. 75 mm.; L. 80. Peroneal muscles, R. 85; L. 84. Gastrocnemius, R. 78; L. 82. Vastus ext., R. 83; L. 80. Interossei, R. 76; L. 80.

*Galvanic Exploration of Muscles*.—Tibialis ant., R. 8.5 ma.; L. 8. K.C.C.=A.C.C.; occasionally A.C.C.>K.C.C. Peroneal muscles, R. 7; L. 7, weak. Contractions normal, rapid. No R.D. Legs thin; atrophic on antero-external aspect.

Taking for our basis this case, and others which we have had the opportunity of examining in the wards of M. Charcot at the Salpêtrière, and of M. Dejerine at Bicêtre, and comparing them with the cases that have been hitherto published, we shall successively consider Friedreich's disease from the etiological, the symptomatic and the diagnostic point of view. Finally, we shall describe its pathological lesions, which class this affection among the combined scleroses of the spinal cord.

## ETIOLOGY.

This case is distinguished from most of those which have been previously observed, by the absence of the *family* or *hereditary* (generic, as Smith calls it) character, which has for long been considered as the rule in this disease. Indeed, the brother and sister of the patient have not been attacked by the same affection, and if we credit M. Soca, who has deduced the law, from the observations of several authors, among others Gowers <sup>(25)</sup> and Rüttimeyer <sup>(41)</sup>, that the children of the same family become affected almost at the same age, we should be able to say that the patient's brother and sister no longer run any risk of having this affection, since they have both passed the age at which the disease commenced in their brother.

M. Soca formulates this law in the following way:—"Friedreich's disease commences at the same age, within two or three years, in all the members of the family which it attacks."

We shall, however, make some reservations on this point, and before adopting this law, the practical importance of which would be very great, we think that it is expedient to wait for a greater number of cases, and especially greater exactness among authors in fixing the onset of the first symptoms. Mr. Soca says so himself (p. 132):—

"The first phenomena which strike the patient and his friends vary somewhat in different cases, sometimes the onset of the disease dates even from birth. The little patients never learn to walk properly. It is only very late that they walk alone, and then their gait already presents all the characters of ataxy. Let me add that this mode of onset is pretty common."

In reality, the onset of Friedreich's disease is so often so insidious and insensible that the patient and his friends do not trouble about them till long after their appearance, when the weakness of the legs and the ataxy of movement have become sufficiently developed to interfere with walking. If the physician who is at length consulted then questions the parents they will tell him that they have noticed, for several years perhaps, certain signs which should have aroused their



suspicious, but without giving serious attention to them, because they attributed them to idleness and obstinacy on the child's part rather than to disease.

Must we then admit that external conditions, environment, occasional causes, are really without any influence on this disease which would thus fatally attack at the same age the members of a family? We must confess that our knowledge of the etiology of this disease is still so obscure that it is actually impossible to say anything on the subject. We think, however, that Friedreich's disease is no exception to the general laws of heredity, and we believe that one would commit a great error in wishing to withdraw it from external influences (always necessary for the development of hereditary morbid germs slumbering in the organism) by reason of the absolute ignorance in which we are of the occasional causes of this disease.

It has been observed in several attacks that the onset of the disease has declared itself after an acute disease—a fall, an injury, or after early habits of onanism. These are the common causes of which we must assuredly take account, but which can only be of influence in individuals hereditarily predisposed.

In the personal history of patients we sometimes find noted scrofula, tuberculosis, and infantile convulsions. But it is the eruptive fevers and typhoid fever which appear to have most often been the occasion of the onset of the weakness of the legs and the ataxy of the extremities. In one of Rüttimeyer's patients the first symptoms of ataxy were manifested during a long convalescence following scarlatina of four weeks' duration. Vizioli<sup>(53)</sup> has seen two cases of Friedreich's disease develop after scarlatina. Ormerod<sup>(54)</sup> also quotes a similar case. Whooping-cough and measles have also been observed to have preceded, by a longer or shorter period, the first manifestations of the disease. Rüttimeyer and Vizioli have each seen a case in which the ataxy of Friedreich appeared after typhoid fever. Let us further note as antecedents the mention of febrile affections not attended by eruptive or cerebral manifestations.

Musso<sup>(49)</sup> insists at some length on small-pox as a



predisposing cause of the disease. He quotes numerous examples of affections of the nervous system observed after small-pox by Gubler, Westphal, Leyden, Bernhardt, Seppilli, &c., and thinks that in three of his patients it is the small-pox which has kindled the disease, which would otherwise perhaps have remained latent all through life. But in examining Musso's case more closely, it may justly be asked if the first symptoms of ataxy should really be attributed to small-pox. Indeed, in one case, the ataxy did not appear till two years after, in another two years and a half, and in the last not till three years after a mild small-pox.

It is to be remembered that our patient experienced a notable aggravation of his disease after an attack of acute articular rheumatism. At the end of convalescence from that disease he could no longer walk alone, as he could previously with the aid of a stick. In Friedreich's seventh case there is an analogous circumstance. Louise Schulz, whose disease commenced at the age of thirteen, was attacked at fourteen with acute articular rheumatism, after which the shaking of the limbs greatly increased, and speech became indistinct and stammering, which was not the case previously.

In other patients chorea has been mentioned as a pathological antecedent. Thus Rütimeyer (<sup>41</sup>) says that Jacob Blattner (his seventh case) had at the age of four years St. Vitus's dance for six months, and that after that affection he remained feeble in his legs and uncertain in his gait; since then, too, the disease invaded successively the upper extremities and speech, as in his eldest sister. We ask ourselves if this chorea should not rather be considered as a symptom of the onset of Friedreich's disease. We have seen that our patient was regarded for three years as suffering from chorea, and we know now that "choreiform movements" form part of the regular procession of symptoms of this disease. We think that chorea should henceforth be considered as belonging to the first period of Friedreich's disease, whenever the latter will appear to be the direct consequence of it. Other concomitant symptoms, such as the abolition of the knee reflexes, will serve in a doubtful case to settle the diagnosis.

Let us relate yet a negative etiological fact, which assumes a certain importance in the enumeration of the causes of so-called hereditary ataxy. We hardly ever find syphilis mentioned in the personal or hereditary antecedents of Friedreich's ataxics—contrary to what is observed so frequently in true tabetics.

On the other hand, alcoholic excess in the parents has been pretty often blamed. Friedreich was the first to insist on this point. The first six cases that he observed belonged to two families, Lotsch and Suss. Now, the fathers of these two families were drunkards, and the widow of Suss said that all her children were conceived during drunkenness. Other observers, notably Everett Smith, Quinke\* and Rüttimeyer have since related analogous examples. There are other cases, as a set-off, where alcoholic excess of the parents is absolutely excluded.

Direct heredity has, therefore, some share in the symptomatology of Friedreich's disease, as in that of myopathic atrophy and Thomsen's disease. There are, however, numerous exceptions to this rule, and isolated cases like the present one are to-day not rare in science. It would not be necessary to think on this account that these last cases escape the law of heredity. We establish most often, as in our patient, amongst ancestors and relatives nervous and psychical affections of diverse nature, so that these sporadic cases fall like the others under the general law of neuro-pathic heredity, although they do not present the "family" character, which is the index of an accumulated converging hereditary influence, that is to say, raised to higher power. (See subjoined the genealogical tables of Rüttimeyer, Musso, Vizioli and Gowers.)

RÜTIMEYER (*Virchow's Archives*, 1883, vol. 91, p. 116 and 117). Blattner "Stulzi" (that is to say, "hobbling") married in 1710.

All the direct descendants of this Blattner are entered under this surname "Stülzi," in the official registers of the commune of Küttingen up till the "forties" of the present century, and the strange gait of this man must have strongly impressed his con-

\* QUINCKE, in Friedreich's second paper, *Virchow's Archiv*, Bd. lxxviii, p. 165, 1876.

temporaries for it to have remained during so many generations in the recollection of the inhabitants of the country. However, the eight cases of Freidreich's disease have only quite recently shown themselves in this Blattner family, whilst previously neither nervous nor mental diseases were known in it.

These eight cases belong to four different branches, of which the great grandfathers were brothers and lived at the commencement of the present century, direct descendants of the famous Blattner-Stülzi. Here are the four branches (in the fourth generation probably, and not in the second, as Soca erroneously states).

#### BLATTNER-BLATTNER.

Ten children, of whom seven healthy.

Rudolf (1). Born 1865. ATAXIC (scarlatinal).

Gottlieb. Born 1848. ATAXIC.

Marie. Born 1853. ATAXIC.

#### BLATTNER-BASLER.

Seven children, six healthy.

Karl. ATAXIC. Mother affected with chorea in childhood. Maternal uncle paralysed in both legs suddenly (acute poliomyelitis).

#### BLATTNER-KYBURZ.

Seven children, six healthy.

RUDOLF (2). Born 1867. ATAXIC (following typhus).

#### BLATTNER-WEHRLI.

Ten children, seven healthy.

Marie Madeleine, age 20. ATAXIC.

Jacob. Died, aged 18. ATAXIC.

Fritz. Died of measles, aged 9. ATAXIC.

As for age and sex, we have but little to say of them. Friedreich thought that hereditary ataxy was a disease commencing at puberty, especially in girls. This observation is not always verified. Many cases date from infancy as well in boys as in girls. After the age of twenty it is extremely rare for Friedreich's disease to show itself.

### SYMPTOMATOLOGY.

The clinical history of our case furnishes a typical picture of the symptomatology of Friedreich's disease, which may thus be recapitulated: slow and progressive ataxy of the four limbs—usually attacking several children of the same family, dating often from very early age—commencing in the legs, extending gradually to the trunk and arms, the muscles of the larynx, those of the tongue and of the eyes; weakness of legs, increasingly difficult gait (tabeto-cerebellar, Charcot); choreiform unsteadiness; static ataxy (Friedreich); difficulty of articulation; nystagmus; spinal curvature; paralytic club-foot; abolition of knee-reflexes; no sensory disturbances; absence of oculo-pupillary anomalies and of lightning pains; integrity of sphincters.

Sometimes other symptoms are added which are due to

complications, or which result from an error of diagnosis. It appears to us necessary from this point of view to eliminate a certain number of cases which are still classed under Friedreich's disease, although they have no right to be so considered. In reckoning these doubtful cases the symptomatology of this affection is confused, and elements of error are introduced which obscure the clinical picture of it.

Let us rapidly pass in review some of the cases which in our opinion ought no longer to figure in the category of Friedreich's ataxias.

There are first of all three patients of Hitzig,\* referred to by authors and hawked about from one bibliography to another without any verification of the reference. As M. Soca remarks, this reference is inexact. It refers in fact to page 513 of the *Berliner Klinische Wochenschrift* of 1875, which is devoted to advertisements! In reality, it does not relate to a work of Professor Hitzig on Friedreich's disease, but simply to a discussion which took place at the Berlin Society of Medicine on January 20th, 1875, after a communication by Dr. Bernhardt on muscular atrophy. Hitzig said that he had had under observation for several months two brothers and a sister affected with muscular atrophy, in whom the disease had existed between five and ten years; seven other children of the same family perfectly healthy. The *mother* of these ten children was *tubetic* in a high degree. But there is not a word about difficulty of walking, which should have been observed in the little patients.

The error proceeds from Seeligmüller,† who thought that the atrophies of Hitzig ought to be classed under Friedreich's disease, perhaps on account of the *tabes* of the mother, and who speaks of difficulty of walking (*Gehstörungen*, loc. cit. p. 188), whereas there is no mention of it in the report of the discussion.

Seeligmüller, moreover, is not fortunate in the choice of observations on which he relies for the description of Fried-

\* *Berliner klinische Wochenschrift*, 1875, p. 143.

† V. SEELIGMÜLLER in Halle, *Krankheiten des Nervensystems* II. *Scleroses der Hinterstränge*, p. 185.—In GERHARDT's *Handbuch der Kinderkrankheiten*, vol. v., part ii., first edition, Tübingen, 1880.

reich's disease. Beyond the cases of Friedreich—the only really authentic ones at that period—he quotes *Du Castel*,\* whose patient was not ataxic but paralysed; the two cases of Kellog<sup>(8)</sup>, too incompletely described and presenting too many anomalies to be admitted as belonging to Friedreich's ataxy; the cases of *Bouchut*† (in which ataxy exists in name only), a boy eleven years of age, ill for fifteen days with tingling in the feet, sometimes diplopia with convergent strabismus and slight constipation, and a girl of fourteen years, an old rachitic subject, ill for two years, who walks with a firm but slow step, without trembling and without defect of muscular co-ordination; moreover, “one may tickle in any way the soles of her feet without making them move” (!!) ; lastly, the case of *Kahler* and *Pick* <sup>(15)</sup>, combined sclerosis of the posterior and lateral columns, which is by no means a case of Friedreich's ataxy, the dominant symptom being paraplegia.

Let us add, that to crown his mistake, Seeligmüller <sup>(23)</sup> has published two cases which certainly do not belong to Friedreich's disease; that Friedreich himself, consulted by the author, has refused to recognise them as analogous to his own, but which Seeligmüller persists in spite of Friedreich's authority, and in spite of evidence, in considering as authentic examples of hereditary ataxy with nystagmus. These are the cases:—

Leo von K——, age twenty-six, descended from an old noble family, in the genealogic tree of which neuro-psychopathies abound. Uncertainty of gait at twelve years, at fourteen progressive myopia, from fifteen to sixteen melancholia. Onanism. Sexual perversion. At twenty-one he enlists as a volunteer in the cavalry. He is a bad horseman. He has learned to swim, but he is a bad swimmer. He dances, but he is a bad dancer, for he easily becomes giddy if he turns round in dancing, and if he rides round. Girdle pains. Urinary troubles. Various psychical disorders. What is most striking in his physiognomy is his nystagmus, which the patient can arrest when he wishes,

\* DU CASTEL, Observation de sclérose primitive des cordons de Goll. *Gazette médicale de Paris*, Jan. 17, 1874, No. 3, p. 33.

† BOUCHUT, Ataxie locomotrice et sclérose des cordons postérieurs de la moelle chez les enfants. Signes ophtalmoscopiques. *Gazette des Hôpitaux*, March 31, 1874, No. 38, p. 297.

so much control has he over himself! He is able to arrest the nystagmus when he fixes his look fifty centimetres in front of him. He then feels firm on his feet. The patient is never at rest, but he can always arrest his movements by an effort of will. No locomotor ataxy. With eyes closed he goes pretty straight to a fixed point in front, but his legs are too wide apart and he walks irresolutely. He mounts a chair, &c., without difficulty. Plantar reflex abolished; cremasteric absent on the right side. *Patellar reflexes very exaggerated.*

Alexander von R—, age twenty-eight, married, robust, very strong, but awkward. From childhood a grimacer; shakes his head; spasms; rabbit-like movements of the nose and lips. Syphilis. Gonorrhœa; vesical catarrh followed by violent pains in the right hip. For six months he has had difficulty in walking (October, 1878), yet he has walked for several hours—limping rather—to go to the chase. Asymmetry of face. Left strabismus. No nystagmus. Speaks very quickly, and gets flurried. Ataxy of thought, but no trace of ataxy of speech. No spinal curvature. Walks with his legs apart, and with swaying of the body. His eldest daughter has the same gait. When upright, with eyes open, the patient does not stumble, nor does he if his eyes are closed. No trace of ataxy in the upper limbs. Plantar and cremasteric reflexes abolished. Knee reflexes very lively.

A sister, whom Seeligmüller was not allowed to examine, suffered, it appears, from symptoms in all respects like those of her elder brothers.

It is indeed sufficient to enumerate these symptoms to understand that Friedreich had good reason not to find in them an image of the disease which he had so conscientiously studied and described. Nor is Seeligmüller's indignant exclamation in any way justifiable, when he says (*loc. cit.*, p. 242): "To conclude, I must protest strongly against the assumption of Bourneville, who argues that the hereditary ataxy of Friedreich is a disseminated sclerosis, with predominant lesion of the posterior columns. The clinical picture offered by the patients of Friedreich, two of whose ataxies I have myself seen at Heidelberg in the autumn of 1878, as by my own is absolutely different from that of disseminated sclerosis."

Who would have doubted that Seeligmüller had seen Friedreich's patients? Doubtless Bourneville was deceived,

but we must confess that nothing resembles more a patient affected with disseminated sclerosis than an ataxic of Friedreich, with his choreiform unsteadiness, his nystagmus and his slow and scanned speech. It seems quite natural that one may have supposed, in 1869, at the time when disseminated sclerosis had just been discovered, and was not yet known under all its clinical varieties, that the first cases of Friedreich might be classed under that disease. We have already seen how Friedreich has replied to it; we shall not return to it.

*Hammond* <sup>(28)</sup> has enriched the statistics of hereditary ataxy by twelve cases, of which the very incomplete, superficial and aphoristic observations do not permit us in the case of any of them to subscribe without reserve to the diagnosis. It is absolutely certain, on the other hand, that most of these cases do not belong to Friedreich's disease. What can the following cases, for example, have to do with this affection? In the family of R—— there are three brothers who died young, and a sister in good health, of whose heredity *Hammond* says nothing.

1. A little boy, G. R——, ill since dentition, general paralysis and articulatory trouble; no pains; sensibility always normal; died aged five years.

2. His brother, L. R——, paralysed at six months; paralysis gradually increasing; died aged three years.

3. A third brother, W. R——, died aged three years; paralysis and articulatory difficulty, without atrophy and anæsthesia.

And authors would regard these patients as cases of Friedreich's ataxy! The twelve cases of *Hammond* should henceforth be erased from the list of this disease.

As much may be said for the two cases of Drs. *Oxley* and *Pollard* observed at the Liverpool Children's Hospital, and reported by *Davidson* <sup>(31)</sup>.

CASE 1. (*Oxley*).—M. B——, girl, aged nine years. Six weeks ago she was attacked without known cause, in two or three days, with muscular inco-ordination of the four limbs, as if she had chorea. Mastication and deglutition difficult. Speech embarrassed. Urine in the bed. Patellar reflexes exaggerated. Sensibility normal. Rapid improvement by appropriate hygiene and good nourishment. After five to six weeks she could walk alone.



CASE 2. (Pollard).—J. G——, little boy, aged seven years. Ten weeks previously an attack of rheumatic fever; for the last eight weeks has entirely lost speech and the use of his legs. At rest in bed no involuntary movements. As soon as he wishes to make a movement, the latter is exaggerated and violent as in chorea. For a reply he makes a motion with his head. Sphincters normal. No exaggeration of tendon reflexes. Sensibility normal. Six weeks after entering the hospital he could walk alone, and stand with his eyes closed. Speech unimpaired.

It must be said that Dr. Pollard does not consider this patient as an ataxic of Friedreich, but considers him to be affected with paresis and functional ataxy, recalling certain hysterical manifestations.

The custom which has been followed, since Seeligmüller, of considering exaggerated or simply normal knee reflexes as belonging to the symptomatology of Friedreich's tabes, has contributed largely to lead astray the authors who have considered under the heading of this disease a number of cases with exaggeration of the knee reflexes, which ought not to figure in it.

Seeligmüller recognises that the symptoms of his patients differ from those which had been given by Friedreich as characteristic of his disease. In his manual he mentions in passing hereditary ataxy in the middle of the description of the symptomatic forms of tabes, and says <sup>(65)</sup>: "The symptoms of the cases observed by other authors and by myself offer a clinical picture more or less different" (*in mehrweniger ausgesprochener Weise* <sup>1)</sup>) to those of Friedreich.

I shall yet quote the two patients of *Massalongo* <sup>(48)</sup>, the one of *Descroizilles* <sup>(68)</sup>, and the one of *Dalché*,\* which certainly do not belong to Friedreich's ataxy. The abolition of patellar reflexes is an absolute rule in this disease, and one will do right always to mistrust the diagnosis of this disease whenever the knee reflexes will not be lost, although all the other symptoms would seem to confirm it.

Another category of patients, on the other hand, in whom the tendon reflexes are abolished, have also been improperly reckoned among the cases of Friedreich's disease, and should

\* P. DALCHÉ, *Maladie de Friedreich. Pseudo-tabes. Discussion. Progrès médical*, June 30, 1888, p. 507.



no longer be classed under it. The anomalies which they present suffice to remove them from this affection, and to place them either under disseminated sclerosis, under the combined scleroses, or amongst other spinal or general nervous diseases.

*Brousse's* patient, for example <sup>(34)</sup>, whose first symptoms, weakness and inco-ordination of the legs appeared only at the age of twenty-four years, had at the end of his life apoplectic attacks which do not form any part of Friedreich's disease. Moreover, the ataxy and the articulatory troubles have peculiar characters foreign to this affection. This is how M. Brousse expresses himself:

"Walking is not possible without assistance. When it is attempted it causes trembling of the whole body. . . .

"When the patient is at rest, if he is questioned rather suddenly his head is affected with trembling. . . . There is difficulty in his speech, it is hesitating, stammering."

The autopsy revealed a combined sclerosis, fasciculated of the posterior columns, and diffuse of the antero-lateral, with central myelitis. It was not therefore Friedreich's disease.

The cases of *Erlenmeyer* <sup>(40)</sup>, *Hollis* <sup>(26)</sup>, *D'Arcy Power* <sup>(32)</sup>, *Fazio* <sup>(56)</sup>, and many others besides, are doubtless liable to the same objections. The case of *Carre* <sup>(2)</sup>, too, offers certain symptoms which render it suspicious. The disease began at twenty-two years by pains in the sole of the feet and in the thigh, then on the inner side of the hands, which were *dead* (sensibility blunted). Diplopia and weakness of sight. Speech impeded; a kind of stammering; one would say that the tongue, hindered by an obstacle, has difficulty in detaching itself from the palate, *then a series of words succeed one another rapidly*—all that is absolutely foreign to the symptomatology of Friedreich's disease.\*

\* I had written this critical review of cases of Friedreich's disease before reading the excellent article which Professor F. Raymond has devoted to *tabes dorsalis* in the *Dictionary of Dechambre* (See *Bibl.*, No. 62). I see with pleasure that M. Raymond no longer admits the following cases as examples of the disease described by Friedreich—(1) the case of *Kahler and Pick* <sup>(15)</sup>; (2) the two cases of *Seeligmüller* reported above; (3) the case of *Brousse*; (4) *Hammond's* case. The author remarks, moreover, that *Carre's*

After throwing out many cases considered by different authors as belonging to Friedreich's disease, we propose, as a set off, to add one which appears to us truly typical. We found it in the work of Topinard<sup>(4)</sup>, and as it has passed entirely unnoticed, we give of it the following *résumé*:

Case 215.—Eldouard L—, aged twenty-one years, tiler. Father and mother in good health. Eight brothers and sisters, of whom five have died before the tenth year. Scrofula; onanism from the age of thirteen, afterwards venereal excess. Periodic migraine every fortnight. Towards the age of eleven years, at la Pitié, M. Michon proposed an operation to remedy a sinking of the left scapula. Towards the age of seventeen, vertigo and cramps. His legs supported him with difficulty; tendency to fall backwards. His speech became difficult. He became humpbacked. Blows with a stick on the back, followed by dull pains for a month. To sum up, during two or three years the above-described phenomena; failures of equilibration, difficulty of pronunciation and gradual deviation of the trunk constituted the whole disease and became more and more pronounced.

Intellectual power excellent; sight and hearing excellent. Neither strabismus nor diplopia. Since the invasion of his disease, no headache, loss of consciousness, nor convulsions. All the movements of the tongue are freely executed; no indication of paralysis of the soft palate. These facts are all the more necessary to note, as the speech is thick, nasal at times, as if the tongue was swollen.

Impressions of cold, of pain, of simple contact, of tickling, as perceived as usual in the face, the trunk, the arms, the legs, the soles of the feet. Muscular sense everywhere unimpaired. Neither numbness, nor tingling, nor pains of any sort. No trembling. Muscular strength very considerable everywhere.

For some months L. has been awkward in his hands, and has allowed objects to fall. When he has his eyes closed he is unable to carry his spoon straight to his mouth, or the point of his index finger to the tip of his nose. In the lower limbs analogous

case<sup>(2)</sup> and Teissier's<sup>(4)</sup> present features foreign to Friedreich's disease, and ends by saying that an attentive reading of most of the cases which it has been sought to connect with Friedreich's disease shows that authors like Bourneville may have wished to see in this disease a defaced form of disseminated sclerosis. Judson Bury (69, p. 20), thinks too that the cases of Seeligmüller belong rather to disseminated sclerosis, by reason of the trembling, of the exaggeration of the patellar reflexes, and of the nystagmus which they present.

symptoms. In bed, he can execute all movements. . . . With eyes open and without any assistance, even a stick, he can go about all day and can ascend or descend stairs. His legs feel stiff, he says, but not weak. Exercise does not tire him. But from time to time he loses his equilibrium, and would fall if he did not find a support within reach.

When his eyes are closed the difficulty of maintaining equilibrium is more conspicuous. He separates his feet to enlarge his basis of support, and throws his arms about in order to balance himself. His movements are not disorderly. His foot alone, moderately raised, falls heavily on the ground a few inches beyond or within the proper spot. He walks more quickly than he wishes to preserve his centre of gravity. At long intervals, when he is strongly exerting himself, cramps and spasms in his legs and toes. Genital functions, micturition, defæcation, normal.

After having left the hospital some months he entered the Hotel Dieu with increased difficulty of walking, which resembles that of individuals affected with locomotor ataxy. *He would be supposed to be drunk.* He separates his legs widely. Persistence of sensibility in its different manifestations—muscular, cutaneous, and of the mucous surfaces. Left on account of *insubordination*.

Topinard adds (<sup>4</sup>): “MM. Duchenne and Vigla have decided as to the diagnosis in favour of progressive ataxy; we have not the courage to do so. The peculiarities of gait in this case differ too much from what is seen in this disease, and are too identical with those of Case 25 (<sup>4</sup>), (p. 20) (cerebellar tumour), the diagnosis of which is not doubtful, for us not to maintain reserve.”

This reserve was justified. The case without doubt belongs to Friedreich's disease. The youth of the patient, the special characters of the tabeto-cerebellar gait, the spinal curvature, the absence of sensory disturbances, the integrity of the sphincters, the articulatory difficulty, finally the inco-ordination—everything concurs to corroborate this diagnosis.

#### ANALYSIS OF CERTAIN SYMPTOMS.

The symptoms of Friedreich's disease offer certain peculiarities which deserve to be examined more closely. I shall not make an exhaustive study of them; I shall limit myself to remark upon some among them which appear to me the most important from the clinical point of view.

The ataxy has a quite special feature in this disease, and it is the observations of the Professor of Heidelberg that the partisans of the motor theory of ataxy always quote to demonstrate that sensory disturbances are not at all necessary for the genesis of inco-ordination of movements.

One might be perhaps spared the long discussions which have taken place, and which do not appear to be near termination between advocates and opponents of ataxy by sensory disturbances, if one had at the commencement clearly distinguished the cases of Friedreich's disease from those which belong to tabes properly so-called.

In the latter disease, the theory sketched out in 1862 by Charcot and Vulpian\* and developed in the following year by Leyden,† appears to explain pretty clearly the ataxy of tabetics in whom the control of sight supplements at least in part the absence of knowledge of position of limbs, which sensory impressions from the periphery habitually give in the healthy condition. The ataxy is then the result of a lesion in the centrifugal conducting paths, the integrity of which is necessary for the co-ordination of movements. It is a *sensory ataxy*. I shall not pass in review the very serious objections which have been made against this theory, but I will say that it is absolutely insufficient to explain the disorders of movements in Friedreich's disease. There cannot be any question here, in fact, of sensory ataxy. The characters which distinguish the ataxy in this affection are precisely, on the contrary, the absence of disturbances of sensation and of the muscular sense, coinciding with a pronounced and generalised motor inco-ordination.

The following fact, which I have often verified in the patient whose case I was just now reporting, appears to me quite characteristic and capable of furnishing on this question a decisive argument against the sensory origin of ataxy in Friedreich's disease.

I have already related in this case that the patient,

\* CHARCOT et VULPIAN, Sur un cas d'atrophie des cordons postérieurs de la moelle épinière, &c., *Gazette hebdomadaire de méd.*, No. xviii., May 2, 1862, p. 281.

† LEYDEN, Die graue Degeneration de hinteren Rückenmarksstränge, Berlin, 1862.

whether his eyes were closed or not, could not touch at the first attempt the tip of his nose or the lobule of his ear with the index finger of one hand when told to do so. Now what appears to me of fundamental importance in order to judge of the nature of the ataxy in this case is that when once his finger touches his face at a certain distance from the point sought, the patient slowly glided his index to the point indicated, *without leaving the surface of the skin*. In thus drawing his finger over the epidermis he evidently guided himself by sensation to remedy his ataxy. The motor inco-ordination, far from being provoked by sensory disturbances, was on the contrary corrected, thanks to the integrity of the sensory tract.

This character, as well as that of the generalisation of the ataxy, of its propagation from the lower limbs to the muscles of the larynx, of the tongue and of the eyes, prove that the seat of ataxy in Friedreich's disease must be central, placed in the very focus of the centrifugal motor paths of co-ordination of muscular movements—that is to say, in the medullary extension of the cord. It is a *bulbar ataxy*.

Hammond has gone farther. He supposes that the seat of the lesion is in the cerebellum. Most physiologists since Flourens consider the cerebellum as presiding over the functions of co-ordination of movements. Hammond relies on this physiological doctrine to advance the theory that Friedreich's disease has its origin in the cerebellum and the medulla, to be propagated thence gradually to the spinal cord, which is consequently, according to this supposition, secondarily diseased. We shall see, in treating of the pathological anatomy, that the autopsies made hitherto do not allow this view to be adopted. Let us remark, however, from now, that it is contrary to clinical observation, since the symptoms of ataxy are propagated from below upwards, and not from above downwards, as they would be if Hammond's hypothesis had some probability. We have seen, moreover, that the cases of the American Professor do not belong to Friedreich's ataxy, so that the unusual symptoms observed in his patients—vertigo, pains in the back of the

head, &c.—should not be reckoned in the symptomatology of this disease. We shall speak of the choreiform unsteadiness, of the movements of athetosis of the toes, of the articulatory troubles and of the spinal curvature when we shall treat of the diagnosis.

As for *nystagmus*, Friedreich had already made a very profound study of it. He had remarked that the patients had no suspicion at all of their nystagmus, although the oscillations of the ocular globe were very pronounced. Moreover, contrary to what is often observed, the nystagmus in Friedreich's cases was produced when an object was fixed, whilst in the ordinary cases of nystagmus of ophthalmologists, it was then made to disappear, as Professor Seeligmüller has expressly observed in one of his patients. In the case we have described, as we have seen, the nystagmus behaved exactly as Friedreich has described it. We know that the latter writer distinguishes two kinds of ataxy, according as the inco-ordination is observed during movements, or at rest during the continuous contraction of the muscles, which maintains the equilibrium of the body and limbs—locomotor ataxy and static ataxy. The ocular movements also present these two kinds of ataxy.

Nystagmus is a late symptom in Friedreich's disease—one which appears generally several years after the weakness and ataxy of the legs, sometimes even eighteen or twenty years after the onset of the disease. Its importance is great, especially as a diagnostic sign.

The other ocular troubles, so frequent in the tabes of Duchenne, are almost unknown in Friedreich's disease. However, Joffroy<sup>(86)</sup> mentions diplopia and drooping of the upper eyelid, and Mendel<sup>(81)</sup> converging strabismus (double paralysis of the sixth pair). In these two cases the pupillary reactions were normal to light, as well as with convergence and accommodation.

*Deformity of the foot* in Friedreich's disease is a symptom which may exist very early. Rüttimeyer relates that they had observed in the families Blattner and Kern the early prominence of the extensor proprius pollicis, which was considered as a sign of very bad augury; the father also

of the children Kern wrote to the doctor that "each time that this traction of the great toe by a sinew" was observed, the parents lost all hope of seeing their child escape the disease.

Friedreich had already observed deformity of the feet in one of his patients, Charlotte Lotsch (his second case): "The group of dorsal flexors of the foot is weakened," says he (the feet are permanently extended by the action of the calf muscles). "*The toes also are permanently flexed,*" and he adds in his second memoir, when speaking of the same case, fourteen years later, "her feet are in a position of permanent equino-varus."

If, therefore, Friedreich has not given a complete description of all the kinds of club-foot which are observed in this disease, we cannot, however, say with Soca that he did not recognise this sign, and we ought to add, in opposition to the statement of Rüttimeyer, that he has also proved the existence of dorsal flexion of the toes.

When deformity of the foot is complete, it is accompanied by certain characters which it is important to recognise to differentiate it from other deformities of the feet, especially the tabetic club-foot, as we shall do in the chapter on diagnosis, where we shall more particularly study this deformity.

What distinguishes the club-foot in Friedreich's disease is, first of all, the condition of equinus which results from the predominance of the muscles of the calf over the antero-lateral group of leg muscles, weakened, and even somewhat atrophied. There is indeed a certain degree of foot-drop in these patients, as we have verified in the case we have related. Some authors think that the condition of equinus must be attributed to the contraction of the sural triceps, but we do not experience any resistance in the tibio-tarsal articulation when we raise the foot, and the tendo achillis is not stretched unduly, so that we can readily admit retraction of the calf muscles, but surely not their contraction in the ordinary sense of the word. We must recollect, however, that the contracted muscles behave in a quite peculiar way in tabetics whose reflexes are abolished.

Westphal demonstrated in 1879 that spasmodic phe-



nomena disappear in the combined scleroses, when to the lesion of the lateral columns is added that of the posterior columns, as is the case in Friedreich's disease.

The articulations remain flaccid. Debove has recently quoted an example of it in an ataxic affected with cerebral hæmorrhage with secondary degeneration of the pyramidal tract, and manifest contracture of the paralysed limbs. Now in this case the reflexes were abolished and the joints remained lax on the hemiplegic contracted side. Might there be analogous contractures in Friedreich's ataxics? That is possible, and then we should have the explanation, not only of the equinus, but also of the dorsal flexion of the toes and of the prominence of the tendon of the extensor proprius pollicis, which also is shortened. This is the opinion of Rüttimeyer, who explains also by contracture the deviation of the spinal column.

Be it as it may, one could not interpret in a satisfactory manner the club-foot in Friedreich's disease simply by the contractures of certain muscles. The paralytic weakening of the antagonists of the shortened muscles also enters into it largely in part, if indeed it is not the essential cause of it. It may well be that the claw-like character of the toes may be the consequence of paralysis of the interossei, and the excavation of the soles of the feet that of the relaxation of the plantar muscles. There are still, as may be seen, many obscure points in the genesis of this club-foot. The phenomena which are observed in these feet give to this disease a particular stamp, which separates it clearly from ordinary tabes, in which muscular atrophies and arthropathies, which are complications relatively pretty rare, give rise to deformities of the foot of quite another form, and quite another nature.

In Friedreich's disease the club-foot is a usual phenomenon which is, perhaps, in intimate and direct relation with the central lesion of the columns of the spinal cord. In every case the particular form of the foot is absolutely different from that which M. Dejerine\* has recently so well

\* J. DEJERINE, Sur l'atrophie musculaire des ataxiques (névrite motrice périphérique des ataxiques), Paris, 1869, and *Revue de médecine*, Feb., March and April, 1889.



studied in his important work on the peripheral motor neuroses of ataxics, in whom muscular atrophy is presented under the form of equinus with plantar flexion of the toes, and particularly of the great toe—that is to say, in a position precisely the opposite of that which is observed in Friedreich's disease.

The *psychical condition* of Friedreich's ataxics deserves special mention. Some authors think that they always suffer from an arrest of intelligence. The case which we relate proves the contrary. I think it is rather in the temperament that anomalies must be looked for. To several patients great irritability of temper is ascribed. Some are apathetic and indolent, others have a caustic turn of mind. We have already spoken of the impulsive laughter which gives such a singular characteristic to the physiognomy.

#### DIAGNOSIS.

The clinical study which we have just made of the etiology and the symptomatology of Friedreich's disease allows us to simplify very much the discussion of its diagnosis.

In the present state of our knowledge we cannot admit indeed, as belonging to this disease, either the cases with exaggeration of the tendon reflexes (Seeligmüller, &c.), or the forms with paralysis and contractures (Soca). M. Soca says that Friedreich's disease can appear under three different forms, according to the nature of the motor disturbances, which may be of three kinds: 1st, ataxy; 2nd, paralysis; 3rd, contracture. Clinical observation does not, however, authorise us to distinguish these hypothetical classes. M. Soca himself admits doubts on the form with contractures, and in order to prove the existence of the paralytic form he is reduced to quote some cases of Hammond and the case of Kahler and Pick. We have already given the reasons on account of which we could not consider these cases as belonging to Friedreich's disease. We know that this disease presents itself clinically as a well-defined type of ataxy dating from childhood, and before placing under its

title other forms foreign to this type, we must evidently wait till the study of the combined scleroses of the spinal cord is more advanced, which will, perhaps, permit us to connect with Friedreich's disease certain unusual cases which it would be at least premature to actually class in it.

In keeping, therefore, to the clinical type which we have learned to recognise in the preceding chapter, we shall say that the diagnosis of Friedreich's disease is based upon the following characters :

Ataxy and weakness of the legs developing gradually in childhood, almost always without pains, but often simulating choreiform movements. Very slow progressive course, from below upwards, of the disease, which attacks successively the trunk, the arms, the muscles of the larynx, of the tongue, and of the eyes. Ataxic and trembling gait, becoming gradually aggravated. No remissions.

Speech slow, drawling, scanned, as in disseminated sclerosis. Static and dynamic nystagmus. Abolition of knee reflexes. Scoliosis. Peculiar deformity of the foot.

To which must be added negative symptoms which are of very great importance :

Absence of lightning pains. Integrity of cutaneous sensation in all its manifestations. Normal reaction of pupils. Integrity of sight. Absence of genito-urinary troubles.

No syphilitic antecedents. *Family* character of the disease. These are the two principal etiological conditions.

The differential diagnosis will bear essentially on the following affections :—

1. Chorea.
2. Tabes.
3. Disseminated sclerosis.
4. The other combined scleroses of the spinal cord.

## I.

Friedreich's disease can be confounded with *ordinary chorea* only at its onset, before the ataxic movements have become sufficiently developed in the lower limbs to interfere with walking. This error of diagnosis, we have already

said, has been made in several cases. We must not, therefore, lose sight of the possibility of a similar mistake, and we shall take care in the cases of chorea which appear suspicious by their manner of walking and their duration, to examine the nature of the ataxic choreiform movements, which are never unilateral in Friedreich's disease, and generally do not invade the facial muscles. We shall assure ourselves also of the presence or the absence of the tendon reflexes, and we shall not neglect to examine the feet, for one of the first signs of Friedreich's disease may be, as we have seen, the dorsal flexion of the great toe, with prominence under the skin of the tendon of its proper extensor.

## II.

*Locomotor ataxy* and Friedreich's disease are distinguished one from another by the most decided characters. Tabes is an affection of adult age; Friedreich's ataxy a disease of childhood. It would not, however, be necessary to go so far as to say with M. Soca: "A child or a youth, who is ataxic is a case of Friedreich's disease. This is not absolutely true, but it is sufficiently exact for practical purposes."

Although tabes of adolescents may be a rare disease, there exists, however, now a sufficient number of authentic cases to invalidate this proposition. On the contrary, it is very necessary to guard against making a like diagnosis; and before admitting that an ataxic child may be affected with Friedreich's disease, it is necessary to search with much care for the presence of the symptoms which characterise this disease. We know that all kinds of cases of infantile ataxy have been too readily attributed to Friedreich's disease, without selection and without criticism, at the risk of spoiling the clinical picture of it.

The tabes of children, if we are to judge of it by the cases which have been published, does not differ from tabes in the adult. The ocular symptoms, the sensory disturbances, the lightning pains, the paræsthesiæ, the gastric crises, and the

urinary troubles, furnish sufficient differential characters to prevent any hesitation in the diagnosis. The importance of syphilis as an etiological antecedent has quite recently been emphasised by a case of Professor Strümpell at Erlangen. It relates to a little girl with hereditary syphilis, who was affected with tabes and general paralysis at the age of thirteen years.\* The same holds good in the case of the little patients of E. Remak,† who suffered from hereditary syphilis, and all three presented atrophy of the optic nerve without ataxy. Dr. Berbez,‡ who relates fourteen cases, concludes that in early tabetics, heredity is strong, and that intensity of the disease and multiplicity of symptoms are the necessary accompaniments of early tabes. He remarks also after the teaching of M. Charcot, that, although essentially hereditary, early ataxy has nothing in common with the hereditary ataxy of Friedreich.

MM. Gombault and Mallet§ have quite recently published a case of tabes occurring in childhood, with an autopsy, the report of which appears to us particularly instructive for the discussion of the differential diagnosis of Friedreich's disease and locomotor ataxy. Here is a *résumé* of this report.

Man, aged fifty-seven, infirm from childhood, died a few months after entering the hospital. The patient has, therefore, been only a very short time under observation at the close of his life. Inquiries as to etiology are almost entirely wanting. His father died when the child was seven years old and was already infirm. There is no information as to him. His mother died of acute mania. The patient was placed in a hospital because, it was said, he was permanently insane, and refused to take food. All that we can learn of his history is that the onset of his disease dated from childhood, that he was not seven years old when he fell ill, and that at ten years of age he was definitely admitted as

\* A. STRÜMPELL. Progressive Paralyse mit Tabes bei einem 13 jährigen Mädchen. *Neurologisches Centralblatt*, March 1, 1888, no. v., p. 122.

† E. REMAK, Drei Fälle von Tabes im Kindesalter. *Berliner klinische Wochenschrift*, 1885, no. vii., p. 105.

‡ P. BERBEZ, Tabes précoce et hérédité nerveuse. *Progrès médical*, no. xxx., July 23, 1887, p. 59, cf. W. JACUBOWITSCH, Tabes dorsalis im Kindesalter. *Archiv für Kinderheilkunde*, V Band, 1884, p. 187.

§ ALBERT GOMBAULT et MALLET. Un cas de tabes ayant débuté dans l'enfance. Autopsie. *Archives de médecine expérimentale*, first year. no. iii., May, 1889, p. 385.

a patient in an asylum. From that period continuous and progressive course of the disease, which consists principally in disturbance of the voluntary movements, involving the four limbs. The patient could not maintain the erect position unless he held on to the furniture, and kept looking at his feet. He fell if his eyes were closed. At the time of admission to the infirmary flaccid paralysis of the lower limbs; absence of tendon reflexes; motor inco-ordination of the upper extremities; inability to appreciate the position of the limbs; muscular atrophy; muscles of the thigh and of the leg altogether atrophied; ape-like hands; pronounced scoliosis, the date of development of which cannot be stated, because the patient does not remember it. Did it exist in childhood? Diplopia; slight nystagmus; profound disturbance of sensibility in all the limbs; manifest retardation of tactile and painful sensations; anæsthesia; analgesia; thermo-anæsthesia; sensibility is intact only in the face; no trembling; no brain disturbance; no incontinence of urine or of fæces; pains from time to time, not definitely of the lightning character.

Talipes equino-varus of the bedridden; white and soft œdema of the malleoli; speech distinct and easy; no hallucination; penis and testicles fully developed; scar on the sacrum; death.

*Autopsy.*—Calcareous plates in the arachnoid, all along the cord at its posterior part, especially in the dorso-lumbar region. Issuing from the lumbar enlargement, the posterior roots are bulky, translucent, gelatinous, nodulated, covered with an abundant meshwork of vessels. The anterior roots also are very much hypertrophied. Spinal cord altogether bulky; grey condition and gelatinous appearance of the posterior columns. Peripheral nerves bulky without nodosities, vascular, rose-coloured, translucent.

*The grey matter is much reduced in size, atrophied, and its shape has altered.* It is thin and delicate in the lumbar region, which one would take at first sight for a section of the cervical region. Antero-lateral columns relatively large. They are furrowed by connected perivascular tracts larger than in the normal condition. Posterior columns small, and stained of a rose colour by carmine, especially at the inner part of the posterior zones. Sclerosis of the posterior columns. In the posterior cornua diminution, almost disappearance, of the bundles of fibres belonging to the posterior roots. In the anterior cornua diminution in number and atrophy of the large motor cells.

Vesicular column of Clarke normal in the dorsal and dorso-lumbar region. The pia mater is everywhere notably thickened.

In the cervical region, sclerosis of the columns of Goll; the posterior root zones are sclerosed in this region only here and there.

Hypertrophic sclerosis of the peripheral nerves is much more developed than in the spinal roots. An almost complete disappearance of the myeline has been observed in the peripheral nerve-tubes which have been examined.

In the discussion of this case, the authors say that it is hardly necessary to point out the characters which differentiate it from Friedreich's disease. However, when we read the description of symptoms which they give, we cannot help thinking that the patient of MM. Gombault and Mallet presented in the course of his disease several symptoms absolutely analogous to those of Friedreich's disease:—motor inco-ordination commencing in childhood; abolition of tendon reflexes; course of the affection slow, unobtrusive, continuous and progressive, attacking the four limbs, without pains at the commencement; talipes equino-varus. Have we really in this case the club-foot of the bed-ridden, as some authors think? Deformity of the vertebral column:—MM. Gombault and Mallet remark in reference to this deformity, that it frequently accompanies various generalised affections which have commenced in childhood. But we have now to find out the differential diagnostic signs which allow us to distinguish this particular case from Friedreich's disease, and scoliosis is precisely a character common to the two cases, the importance of which must not be minimised. Nystagmus:—it is true that the authors quote the absence of nystagmus (*loc. cit.*, p. 403) as contributing to exclude Friedreich's disease in their case. They have forgotten that some pages before (*loc. cit.*, p. 387) they themselves have expressly mentioned the existence in their patient of a "slight nystagmus." Finally, the integrity of the sphincters: That is quite as many symptoms as one could refer to Friedreich's disease. And when the authors affirm that the complete absence of cerebral symptoms (mental symptoms being necessarily put on one side) bears witness in their case against Friedreich's disease, they forget again that they have mentioned diplopia (*loc.*

*cit.*, p. 387) in the antecedents of their patient. What, then, are these cerebral symptoms which characterise "the now classical description of Friedreich's disease"? It is precisely, on the contrary, in the absence of these symptoms that we must find a character of the first order to distinguish this disease from ordinary tabes. The authors add, it is true, "and in particular, absence of articulatory troubles," showing thus that they include these troubles among the cerebral symptoms. But those with nystagmus are the only really classical bulbar symptoms which belong incontestably to Friedreich's disease!

We must seek elsewhere for the fundamental reasons which do not allow us to admit Friedreich's disease in the case of MM. Gombault and Mallet. They are especially the generalised muscular atrophies, and the intensity and the extent of the sensory disturbances. These are the essential symptoms which absolutely exclude, in this case, Friedreich's disease.

The authors, who found at the autopsy the peripheral nerves more diseased than the spinal roots, put forward the fairly probable hypothesis of the possibility of a polyneuritis followed by an affection of the cord. Perhaps, say they, we have to deal in this case with multiple peripheral primary neuritis, with consecutive involvement of the spinal cord in the course of development. They arrive, however, at the provisional conclusion, "that we must consider the case as an example of locomotor ataxy of spinal origin, developed in early age."

There is a hypothesis which pleases us more, and we have been struck by not seeing it expressed in the interesting work of MM. Gombault and Mallet. We shall willingly admit in this case a syphilitic affection, perhaps congenital. We know how frequent syphilis is in the hereditary antecedents of tabetic children—we have just given several examples of it. It would therefore be quite possible that syphilitic infection may have also played its part in the patient of MM. Gombault and Mallet. We must not forget either that there existed in this case the remains of a very pronounced anterior poliomyelitis, which also complicated the symptomatology of it.



Let us return to the differential diagnosis of locomotor ataxy and Friedreich's disease, and let us prove first that the symptoms which they offer in common, when examined more closely, are not at all alike.

The ataxy itself presents considerable differences in the two diseases, and the footprints show that the gait is far from being identical in tabes and in Friedreich's disease. In the latter affection the patient progresses in a very irregular zig-zag manner, instead of keeping in a straight line as tabetics do. Nothing displays better than the method of taking footprints, the fundamental difference which exists between the clubfoot of Friedreich's disease, and the deformities of the foot in tabes. The tracing of tabetics affected with arthropathies of the feet shows the print of flat feet, whilst that of the clubfoot of Friedreich is characterised by the complete absence of the print of the sole of the foot corresponding to the excavation which we have described. M. Joffroy (86 and 103) judiciously remarks, moreover, that the deformity of the foot is never developed in tabetics, except when the patient has been for some time confined to bed, whilst in Friedreich's disease the deformity of the foot exists in the patient who is able to walk, long before he is confined to bed.

Romberg's sign discloses also, as we have observed, a fundamental difference in the two diseases. Whilst in tabes closure of a patient's eyes makes him lose his equilibrium, because he has no longer knowledge of the position of his limbs, in Friedreich's disease it is in consequence of choreiform instability. The patient sways whether his eyes are opened or closed, but in the latter case having lost the control of vision, the swaying movements easily pass the limits of equilibrium, and he falls, although he may have preserved the notion of the position of his limbs.

Must we consider, with M. Joffroy, diplopia as a symptom that may belong to the picture of Friedreich's disease (103). We should not dare to speak affirmatively on this point. Diplopia, indeed, in M. Joffroy's cases has been transitory. It is noted in the history of the patient, who belonged to a tuberculous family, but it was not directly observed, we



think, in the medical examination. In every case it does not appear to have presented the lasting characters which it sometimes manifests in tabes. Transitory diplopia is met with in a great number of nervous affections. It is not rare in certain neurasthenics. It has been several times seen in cases of poisoning by tainted meat. Prof. Bouchard\* and many other authors have pointed it out in dilatation of the stomach, in which we have many times proved it. It would not, therefore, be right to conclude from its presence in the history of a patient suffering from Friedreich's ataxy that it necessarily forms a part of the assemblage of symptoms in this affection. We must wait for further observations before admitting diplopia as a definite symptom of Friedreich's disease, of the same importance as in locomotor ataxy. And even, if it ever is admitted as such, it will not remain less exceptional in the first, whilst it is usual in tabes.

We shall not insist any further on the differential diagnosis of these two diseases, which is now well known, and does not offer any difficulty in ordinary cases.

A word only on nystagmus, very frequent as is known in Friedreich's patients, and which was thought never to exist in tabetics. It is observed, however, very clearly in some cases of tabes, but it is true that these cases are very rare. M. Gilles de la Tourette has published three cases of it. I have seen it myself in two cases, one of which is briefly reported in the note on my observations on suspension in tabes.†

In the following comparative table is a résumé of the differences which exist between the two diseases, the symptoms being put parallel :—

FRIEDREICH'S DISEASE.	TABES.
"Family" character. Several children are usually attacked in the same family.	Rarely more than one tabetic in the same family.
Scarcely ever syphilis in the antecedents.	Syphilis is usually found in the history, either acquired or hereditary.

\* CH. BOUCHARD, *Leçons sur les auto-intoxications dans les maladies*. Paris, 1887, p. 174.

† LADAME, *De la suspension dans le tabes*. *Rev. méd. de la Suisse rom.*, June 20, 1889, p. 347.

The disease begins in childhood. There are no authentic cases known of onset after the 20th year.

No lightning pains either at the onset or during the course of the disease. They have been only exceptionally observed, and in these cases are they really lightning pains?

No preataxic stage. The commencement of the disease may simulate a slight chronic chorea for several years.

Ataxy during rest and during movement. Choreiform instability. Movements inco-ordinate, feeble and not sudden. Ataxy is general. It progresses slowly from below upwards. Gait, tabeto-cerebellar, ataxic and festinating. Closure of eyes causes loss of equilibrium only by reason of the choreiform instability, due to static ataxy.

Integrity of sensation in all its manifestations. Towards the end of the disease only, in some cases, slight affections of the cutaneous sensation.

Absence of knee reflexes. Cutaneous reflexes normal. Plantar reflexes rather exaggerated on account of the great sensibility of the soles of the feet.

Integrity of the special sense organs. Horizontal nystagmus, static and dynamic, is rarely absent. Very rarely affections of the muscular innervation of the eyes.

Absence of visceral affections.

Onset almost always after the 20th year. Infantile tabes is most often of syphilitic origin.

Lightning pains perhaps preceding ataxy for several years. Scarcely ever absent in tabes.

Long preataxic stage, presenting the most diverse symptoms: ocular, visceral, &c.

Ataxy during movement. At rest the patient is quiet. Ataxic movements violent, sudden, as of a spring which is let go. The ataxy may be localised in the legs or in the arms (cervical tabes). Gait plainly tabetic. The patient follows a straight line. Closure of the eyes causes loss of equilibrium, because it takes away the knowledge of the position of the limbs (Romberg's sign).

Anæsthesia and hyperæsthesia patches. Paræsthesia. Retardation of transmission of painful sensations and other perversions of sensation.

Absence of knee reflexes. Cutaneous reflexes often weakened or abolished on account of affections of sensation; the plantar reflexes especially are frequently absent.

Grey induration of the optic nerve. Amaurosis. Sclerosis of the auditory nerve. Buzzings in the ears. Menières' vertigo. Myosis and Argyll-Robertson sign. Pareses of muscles of the eye; diplopia, ptosis. Scarcely ever nystagmus.

Laryngeal crises (spasms of the glottis). Gastric, intestinal, anal, vesical, nephritic, crises.

No lesions of the skin, the joints or the bones. Club-foot of special kind, habitual, in consequence of pareses and muscular contractures. Athetosis movement of the toes in walking. No peripheral neuritis.

Integrity of the genito-urinary functions and of the sphinters.

Scoliosis.

Speech slow, scanned.

Progressive, uniform, very slow development of the disease, without crises or prominent episodes.

Cutaneous lesions (zona). Trophic affections of the nails. Shedding of the teeth. Spontaneous fractures. Arthropathies. Tabetic foot. Muscular atrophies. Peripheral neuritis, sensory and motor. Other trophic troubles. Perforating ulcer of the foot.

Genito-urinary troubles. Impotence. Paresis and hyperæsthesia of the bladder. Retention and incontinence of urine. Constipation.

No spinal curvature.

No speech troubles.

Pretty often relatively rapid development of the disease by successive crises, followed by obvious aggravation, with numerous fresh symptoms from time to time.

### III.

In the memoir of Marie,† and in the more recent one of Unger,\* will be found very complete bibliographic notices on *disseminated sclerosis* in children, to which we shall add the work of Professor D'Espine, of Geneva.† It is seen from these works that disseminated sclerosis most often presents in children the cerebro-spinal type which commences by convulsions. The only positive symptoms which may be common to this affection and to Friedreich's disease are the nystagmus and the slow and scanned speech. But these troubles are late in Friedreich's disease, and only appear long after the ataxy, the loss of knee reflexes and the other symptoms which cannot leave any doubt as to the diagnosis,

† PIERRE MARIE, De la sclérose en plaques chez les enfants. *Revue de Médecine*, 1883, p. 536. See also MONCORVO, Contribution à l'étude de la sclérose multiloculaire chez les enfants, Paris, 1884.

\* L. UNGER in Wien. Ueber multiple inselförmige Sclerose des Centralnervensystems im Kindesalter, Leipzig und Wien, 1887.

† A. D'ESPINE, Deux formes de paralysies chez les enfants. *Revue médicale de la Suisse romande*, March 20, 1889, No. 3, p. 129. See also D'ESPINE et PICOT, Manuel des mal. de l'enf., fourth edition, p. 441.

whilst the exaggeration of the reflexes, the rigidity of the legs, the contractures, the trembling on voluntary movement, the disturbances of the motor innervation of the eyes, diplopia, nystagmus, strabismus, &c., are the first signs of disseminated sclerosis. Moreover, all the other symptoms so characteristic of disseminated sclerosis, apoplectiform and epileptiform attacks, vesical paralysis, trophic and mental disturbances, frequent remissions in the course of the disease, all these are invariably wanting in Friedreich's disease, as this author himself had already plainly stated.

#### FRIEDREICH'S DISEASE.

*Family* character of the disease. Insidious onset.

Very slowly progressive course of the affection. No remission of symptoms.

Choreiform instability. Athetosis movements of the toes. Paresis of legs, with loss of tendon reflexes. Progressive ataxy of the four limbs.

Tabeto-cerebellar gait. Integrity of special sense organs and of the ocular muscles. Late nystagmus.

Scoliosis. Special club-foot. integrity of genito-urinary organs and of the sphincters. No visceral tabetic troubles.

#### DISSEMINATED SCLEROSIS.

Case isolated in a family. Sudden onset, often with convulsions.

Frequent remission of symptoms. Irregular course of the disease.

Trembling on voluntary movement. Spastic paresis. Exaggeration of tendon reflexes. Ankle-clonus. Contractures.

Spastic gait. Paralysis of ocular muscles. Diplopia. Amblyopia. Early nystagmus.

Congestive, apoplectiform and epileptiform attacks. Vesical paresis and other tabetic symptoms, generally slightly marked. Mental disturbances.

#### *Characters Common to the two Diseases.*

Absence of sensory disturbances, slowness of speech, scanning of words. Nystagmus.

Vertigo has also been noted as a symptom common to the two diseases, but we do not yet know if vertigo really belongs to this affection, although it may often have been mentioned in reports, since the first case described by Friedreich. Our patient has never experienced vertigo.

## IV.

*Combined Sclerosis of the Spinal Cord.*† We shall not undertake here the study of the subject of the combined sclerosis, which is, perhaps, really the most obscure of spinal pathology. From the anatomical point of view, Friedreich's disease is a combined system sclerosis of the posterior and lateral columns, which could not, in many cases, be distinguished, as regards its spinal localisation, from other combined sclerosis, having a like anatomical seat, with very different clinical symptoms. It is evident that the special evolution of the lesions, their variable extent and the successive separate invasion of different systems of fibres, have a decisive importance for the symptomatology. Friedreich's disease which evolves on an incompletely developed cord, forms a very distinct group, and henceforth is classed among the combined system sclerosis.\*

In spite of the anatomical lesions which it presents, we yet do not think, with Professor Grasset, that Friedreich's disease can be put in the category of "combined tabes." We think it would be preferable to reserve this appellation for the cases of tabes which are complicated by a diffuse sclerosis of the lateral columns, most often following a posterior spinal meningitis, meningo-myelitis by propagation, as Friedreich and M. Dejerine† have proved it. As for the combined sclerosis properly so-called of the postero-lateral columns, with primary system lesions of the posterior columns and pyramidal tracts, we actually possess a number of cases of them sufficient to sketch out the

† See also J. L. PREVOST, *Sciérose des cordons postérieurs, compliqué d'une sclérose systématique des cordons latéraux*. *Archives de Physiologie*, 1877, t. iv., p. 764. V. BABESIU, *Virchow's Archiv.*, 1879, vol. lxxvi., p. 74. EDDES, *Boston Med. et Surg. Journal*, September 21, 1882. RAYMOND, *Arch. de Physiol.*, 1882, x., p. 456. DAMASCHINO, *Gazette des Hôpitaux*, No. 1, 1883. HOPKINS, *BRAIN*, October, 1883. ORMEROD, *id.* April, 1885. BABINSKI et CHARRIN, *Revue de Méd.*, 1885, November, p. 962. MASSALONGO, *Medic. Contemp.*, October and November, 1886. BORGHERINI, *Riv. sper. d. Freniat.*, XIII., p. 137, 1887, &c., &c.

\* Among recent treatises that of Professor L. HIRT of Breslau (*Pathologie und Therapie der Nervenkrankheiten*, Wien, 1890, p. 364), is to our knowledge the only one in which Friedreich's disease is separated from tabes and placed among primary combined sclerosis.

† DEJERINE, *Archives de Physiologie*, 1884, iv., p. 456; *Semaine Médicale* 1886, No. 18, p. 181.

symptomatology of them. There exist, doubtless, very considerable differences in the symptoms described by observers in the different cases that we know. Nevertheless, we can provisionally distinguish from the clinical point of view, four or five categories of patients affected with combined sclerosis, which belong to the following types (without mentioning syphilitic myelitis and others, which give rise to secondary sclerosis more or less diffused, and which may simulate system sclerosis):—

I. *Ataxic paraplegia* (Gowers†). In this we may have all the usual symptoms of tabes with predominance of paralytic weakness of the legs belonging to sclerosis of the lateral columns. If the lesion of the posterior columns stops in the dorsal region, or even if it descends in the lumbar region without attacking the posterior root zones, the knee reflex will be intact, or even exaggerated, in consequence of the sclerosis of the pyramidal tract (Westphal\*).

II. *Spastic tabes dorsalis* (Charcot, Erb, Strümpell). On several occasions combined sclerosis of the postero-lateral columns has been discovered at the autopsy of patients having presented the clinical picture of spastic spinal paralysis, whenever sclerosis of the pyramidal tracts involved all the lumbar region.

III. *Ataxo-spastic tabes* (Grasset†). A mixture of the symptoms of locomotor ataxy of Duchenne with those of spastic tabes.

IV. *General paralysis*. Westphal has shown that primary sclerosis of the lateral columns combined with system sclerosis of the posterior columns is especially frequent in general paralytics. According to this observer primary

† R. GOWERS, Clinical Lecture on Ataxic Paraplegia. *The Lancet*, 1886, vol. ii., p. 1, 61 and 130.—DANA, Progressive spastic ataxia (combined fascicular sclerosis) and the combined sclerosis of the spinal cord. *The Medical Record of New York*, 1887, July 2, vol. xxxii., No. 1, p. 1. DANA, Ataxic Paraplegia, BRAIN, January, 1889, p. 490.

\* WESTPHAL, *Virchow's Archiv*, Band XXXIX. and XL.; *Archiv. für Psychiatrie*, Band V., p. 803, Bd. VIII., p. 469, Bd. IX., p. 413 and 691, Bd. XV., p. 224. See also STRÜMPELL, Ibidem, Band XI., p. 27, Band XVII. (1886), p. 217. DEBOVE, De l'hémiplégie des ataxiques, *Progrès Médical*, 1881. No. 52, p. 1021, No. 53, p. 1042.

† GRASSET, *Archives de Neurologie*, 1886, t. xi., p. 156 and 380, t. xii., p. 27. Cf. BALLET and MINOR, Ibidem, 1884, t. vii., p. 44.

isolated sclerosis of the lateral columns has hitherto been certainly observed only in general paralysis.†

We have no need to go into the arguments which allow us to make the differential diagnosis between Friedreich's disease and the combined scleroses of the spinal cord which belong to the four preceding groups. This diagnosis can offer no difficulty.

It is not the same with a final group in which may be included all the cases which do not enter into the above categories. But it is here precisely that investigations and authentic cases are wanting. Some facts might raise a suspicion that there exists perhaps a form of Friedreich's disease peculiar to adult age; but we are still reduced on this point to a simple hypothesis which requires confirmation. Under these circumstances there can be but one course to follow. We must keep rigorously to the symptomatology of Friedreich's disease such as we know it, with its hereditary characteristics, and eliminate from it as we have done, all the cases which do not exactly correspond with it. The analogies, still very obscure, of pathological anatomy, ought not to serve us as a guide in these questions, but much rather clinical observation, till a deeper study of the combined scleroses, system or diffused, will better enable us to enlighten ourselves in these delicate and complicated researches of spinal pathology.

The report presented by Dr. Oppenheim at the meeting of the Society of Psychiatry of Berlin, Nov. 12, 1888, is particularly instructive on this point.\* It is a case of combined sclerosis of the spinal cord in a child aged 15 years, who suffered from a progressive affection of sight from the age of 10 years, after measles, but whose other spinal symptoms only appeared at the age of 12 years, after a fall in the water. At first choreiform movements in the left half of

† ZACHER, Beiträge zur Pathologie und pathologischen Anatomie der progressiven Paralyse. *Archiv. für Psychiatric*, Bd. XIV. 1883, p. 463, Bd. XV., 1884, p. 359. SIOLI, Ein Fall von combinirter Erkrankung der Rückenmarkstränge mit Erkrankung der grauen Substanz. *Archiv. für Psych.*, vol. xi., 1881, p. 632.

\* OPPENHEIM, Ueber einen Fall von combinirter Erkrankung der Rückenmarkstränge im Kindesalter. *Neurologisches Centralblatt*, December 1, 1888, No. 23, p. 647.



the body, especially in the face and the left arm, then weakness and slight contracture in the joints of this arm. A second group of symptoms is allied to the clinical picture of tabes. Double optic atrophy. Pupils fixed. Ptosis on the right side. Absence of knee reflexes. Weakness and slight ataxy of the limbs. Romberg's sign. No sensory nor sphincter troubles. Death from bed sore. Sclerous atrophy of pyramidal tracts and of the columns of Goll and Burdach. The lesion of the posterior columns was less marked than that of the lateral columns.

We see from the above description of symptoms how easy it was to exclude Friedreich's disease in this case. Oppenheim's case shows that there exists a combined sclerosis of the postero-lateral columns in children with symptoms in great part tabetic, and essentially different from those of Friedreich's disease. This case would be sufficient to distinguish clearly the latter disease from the combined scleroses, and to give it a place apart in the primary system lesions of the spinal cord, in spite of the anatomo-pathological analogies which bring it near to other combined scleroses.

#### PATHOLOGICAL ANATOMY.

The number of autopsies which have allowed us to fix the anatomical lesions of Friedreich's disease is still very restricted, and if one can say henceforth that these lesions belong to the group of primary combined system scleroses, there yet remains no less obscurity on their nature and their precise localisation.

We have altogether nine autopsies of authentic cases, of which five belong to Friedreich's patients, one to Everett Smith, one to Newton Pitt, and two to Rütimeyer.

*Friedreich* <sup>(11)</sup>, who had himself made the autopsy on four of his patients, arrives at the following conclusions:—

The "hereditary" character of the disease is explained, says he, by an arrest of development of the spinal cord. The volume of this organ was very much reduced in all the cases in which an autopsy was made. The spinal cord was



slender, flattened, and as if atrophied in its entirety. It was not difficult to convince oneself that the bulb, and also the cord, had not reached their complete development, so that the sclerosis degeneration of the posterior and lateral columns had invaded an organ still incompletely developed. This is confirmed by the discovery of Dr. Schultze, who found, on microscopical examination, the nervous elements of the healthy portions of the cord very small and very slender.

At the first autopsy Friedreich saw only sclerosis of the posterior columns. The anterior and lateral columns are noted as healthy. In the three others the invasion of the lateral columns by the sclerosis has been clearly determined. But what especially struck Friedreich is the posterior spinal meningitis, which is the true cause, according to this author, of the propagation of the chronic inflammation from the posterior columns to the lateral columns. That is why, says he, in the lower portions of the cord, where the meningitis is more recent, the margin of the lateral columns is less degenerated than in the cervical portion, where all the periphery is degenerated like a ring. We know now that this explanation of Friedreich has no longer much value. In fact, the difference which he had proved between the extent of the cortical degeneration in the lumbar and cervical regions proceeds from this, that in the latter the direct cerebellar tracts are degenerated; there we have a primary system sclerosis which forms part of the combined lesions of Friedreich's disease, the significance of which was completely ignored at the time of the first autopsies made by this eminent observer.

It is true that in Friedreich's cases, and especially in the one, the autopsy of which was made by Schultze (see later), the cortical degeneration went appreciably beyond the region of the direct cerebellar tracts. But the lesions of the cord were examined in these cases only after the disease had lasted twenty-three and thirty years, whilst in Rüttimeyer's cases, the duration of which was only thirteen and nine years, the lesions were confined, as we shall see, to these tracts. We must therefore suppose that in Friedreich's

cases the sclerosis has extended itself, and has gone beyond its first limits owing to the long duration of the disease, so that the diffuse cortical degenerations may be regarded as accessory, having slowly developed themselves, perhaps under the influence of the slight spinal meningitis, and at length complicating the primary system changes.

The cells of Clarke's columns are in intimate relation with the direct cerebellar tracts, of which they represent the trophic centres. Now Friedreich had already remarked that there existed in one of his cases a double canal in the dorsal region of the cord. This double canal, says he, is probably nothing else than the degeneration of Clarke's columns, a degeneration verified in all the other cases, in which the number of Clarke's cells was very much diminished, sometimes even reduced to a solitary one in the lower dorsal portion, that is to say in the region in which they are usually most developed.

A fifth patient of Friedreich was examined post-mortem by Dr. F. Schultze (<sup>20</sup>), who found lesions similar to the preceding ones: grey degeneration of the posterior columns and of the posterior portions of the lateral columns; flattening of the cord from before backwards; slight thickening of the pia mater. Small size of the healthy portions of the cord. As a difference no circular complete cortical degeneration in the upper cervical region, but degeneration of the inner parts of the anterior columns along the longitudinal fissure extending up to the pyramids. Degeneration of the columns of Clarke. Posterior roots atrophied; integrity of the ganglia of the medulla oblongata.

*Everitt Smith* gives drawings of sections of the spinal cord of his patient, and describes the anatomical lesions as follows:—

Spinal meninges injected and adherent to the bony walls of the canal. The cord presents an asymmetric atrophy, it is pale and softened in places. The dorsal and lumbar regions, carefully examined, show marked sclerosis of the posterior columns in their whole extent, with the exception of a small part, relatively healthy immediately behind the posterior commissure. Sclerosis of the direct and crossed

pyramidal tracts less intense than in the posterior columns. Thickening of the pia mater; atrophy of the posterior roots; the anterior normal. Ganglion cells of the anterior and posterior cornua less numerous than normally, and showing in a high degree degenerative change.

Numerous figures demonstrate the seat and extent of the combined sclerosis in Dr. Pitt's case (<sup>74</sup>), the autopsy of which is described with the most minute details. The author gives a series of comparative measurements of a healthy cord and of the diseased cord, which prove how much smaller the sections of the latter were than those of the other throughout the length of the cord. Posterior roots very slender also, especially in the cervical and lumbar enlargements. Nowhere opacity or thickening of the meninges, which were found completely normal. Grey degeneration of the posterior and lateral columns. The hardened cord was examined by Weigert's method. We shall not reproduce the interesting details which Dr. Pitt gives of each region examined, we shall limit ourselves to presenting a *résumé* of his conclusions (*loc. cit.*, p. 389).

I. The spinal cord is extremely slender.

II. Extreme sclerosis of the columns of Goll in their whole length from the lumbar enlargement to their termination in the floor of the fourth ventricle.

III. Intense sclerosis of the posterior part of the columns of Burdach, in which, however, some healthy fibres are scattered, in greater number at the upper part of the cord.

IV. The zone of the columns of Burdach, which bounds the cornua and the posterior root, is intact. (This region bears now the name of marginal zone of Lissauer).

V. Sclerosis not distinctly limited (much less intense than in the posterior columns) of the crossed pyramidal tracts.

VI. Sclerosis of the ascending cerebellar tracts, visible up to the decussation of the pyramids. Very slight and irregular sclerosis of a few scattered fibres in the antero-lateral columns, especially at the periphery, and in some sections along the anterior fissure.

VII. Degeneration of the columns of Clarke in some sections.

VIII. Degeneration of some fibres of the posterior roots, and of the posterior cornua.

IX. Friability and shrivelling of the affected regions.

What was most striking to the naked eye was the great diminution of the transverse section of the cord, as well marked in the cervical enlargement as in the dorsal region and in the lumbar enlargement, the posterior portions being proportionally more diminished than the anterior.

Columns of Goll extremely sclerosed. Numerous healthy tubules scattered in the columns of Burdach and in the anterior third of the posterior columns. A fasciculus almost intact along the posterior cornua. Sclerosis of the crossed pyramidal tracts, and of the direct cerebellar, less pronounced. A slight annular degeneration extends from the periphery to the anterior part of each side of the anterior fissure.

The most marked sclerosis is always in the posterior columns. Posterior nerve roots in part degenerated. In the lateral columns the sclerosis is limited to the crossed pyramidal tracts, and to the direct cerebellar tracts. No degeneration around the posterior commissure. Clarke's columns are sclerosed.

Posterior columns almost completely sclerosed. Anterior quite normal. The triangular sclerosed patch of the lateral column corresponds exactly with the crossed pyramidal tract in this region. It is separated from the posterior root by a band of healthy tissue (marginal band of Lissauer).

Dr. Pitt remarks finally on the frequency among these patients of cardiac troubles, which have not yet been noticed by authors, who give for the most part little attention to abnormal conditions of the circulation and of the blood-vessels, which are frequently degenerated, according to Pitt, in Friedreich's ataxics.

In favour of an association of Friedreich's disease with a tendency to vascular degeneration and to the imperfect development of the spinal cord, Dr. Pitt draws attention to the following points:—

1. That the posterior columns which are the most vascular, have always been found smaller and more atrophied than the rest of the cord ;

2. That vascular lesions have been demonstrated in several cases, and that the disease has always been much aggravated after acute intercurrent affections ;

3. The development of the cord begins in the most vascular regions, so that the degenerations attack by preference the parts which are developed in the last place. These parts have also the least important functions, and, according to the general law of Hughlings Jackson, these are the first which degenerate ;

4. The sclerosed regions correspond to the tracts, the filaments of which are covered with myeline after the 5th month of foetal life ;

5. The symptoms become more marked at the time of puberty, which is accompanied, as we know, by great vascular modifications in the organism.

The two most recent autopsies are those of Dr. Rüttimeyer, of Bale (<sup>77</sup>). By the courtesy of this distinguished writer I have been able to examine with him the interesting series of sections which he has made in his two cases. I can, therefore, speak with authority, and I embrace this opportunity to address again my thanks to Dr. Rüttimeyer. The following is a résumé of these two autopsies :—

1. H., age twenty years, died rather suddenly of general weakness after 13 years' illness. Medulla oblongata and spinal cord very much atrophied throughout. Thickening of the meninges and multiple adhesions, especially in the lumbar region. Posterior roots very much thinned. Firm consistence. With the naked eye grey degeneration of the column of Goll is very well seen in the upper cervical region, and of the columns of Burdach, and of the posterior portions of the lateral columns in the cervical enlargement and the dorsal region. This degeneration corresponds exactly in the lateral columns with the direct cerebellar tracts and the crossed pyramidal tracts. It is clearly separated from the posterior cornu on either side by a narrow zone of healthy white matter. It is in the lower dorsal region that the sclerosis attains its maximum intensity, as well in the lateral as in the posterior columns. In the upper lumbar

region the middle fibres of the radicular zone are much degenerated, which involves the abolition of the knee reflexes during life, as the researches of Westphal have shewn. Absolute integrity of the marginal zone of Lissauer, and of the grey substance generally; whilst in the dorsal region the columns of Clarke, in the grey matter, are degenerated and their cells atrophied. This atrophy is at its maximum in the lowest dorsal region, where ganglion cells are no longer to be found.

Symmetrical degeneration of the lateral columns, strictly limited to the direct cerebellar and crossed pyramidal tracts. Maximum sclerosis in the medium portions of Goll's columns.

Almost complete degeneration of the posterior root zones. The degeneration of the lateral column corresponds exactly with the section of the crossed pyramidal tract in this region.

2. Girl, age 14, ill for nine years, died of general debility following typhoid symptoms. Spinal cord very slender, especially in the dorsal region. The same symmetrical lesions of degeneration in the posterior and lateral columns (crossed pyramidal and direct cerebellar tracts) as in the first case. The medulla of extremely small dimensions. Microscopic examination, made with the greatest care, as in the foregoing case, reveals the same sclerous degenerations in the different regions of the spinal cord. In the grey matter of the dorsal region the columns of Clarke are much degenerated; also the posterior roots. Moreover, the posterior cornua are inact. The same integrity of the zone of Lissauer as in the first case.

Intense degeneration of the columns of Goll. Degeneration of the columns of Burdach reaches to the neighbourhood of the posterior columns from which it is separated by an intact zone. In the lateral columns the sclerosis involves exactly the section of the direct cerebellar and crossed pyramidal tracts.

The maximum sclerosis is always in the columns of Goll, next in those of Burdach. The degeneration of the lateral columns is less pronounced. A slender band of normal

tissue borders the posterior cornua. The antero-external zone of the posterior columns is normal. The degeneration of the lateral columns extends in front beyond the limit of the cerebellar tracts. Is this an individual anomaly or a marginal cortical sclerosis? Accessory zone of Lissauer absolutely intact.

In these two cases the histological nature of the lesions was identical with that which we meet with in tabes. The atrophied nerve tubes are scattered in a very close meshwork of extremely fine connective fibrils crossing in all directions. Nowhere do we find ectasis of vessels, nor thickenings of the walls of vessels which penetrate from the pia mater into the cord. The diffuse general thickening of the pia mater is but slightly pronounced, and is not at all more marked at the periphery of the cord at the places affected by sclerosis. Contrary to what Friedreich thought, there cannot then be any question here of a propagation of the inflammatory process of posterior lepto-meningitis to the lateral columns.

Rütimeyer has also found some degenerated nervous fibres in the median and in the sciatic, but the degeneration of these nerves was, to sum up, very slight. On the other hand the posterior roots were everywhere degenerated, least so in the cervical, most in the lower dorsal and lumbar regions.

Primary, combined, system sclerosis was very distinct in the columns of Goll, of Burdach, in the crossed pyramidal and the direct cerebellar tracts. The lesion of these diverse systems could be followed from above downwards in the spinal cord from the highest cervical to the sacral region. We refer to the important work of Rütimeyer for the detailed description of these lesions. We shall, however, remark that in the two cases the posterior radicular regions of Westphal, the integrity of which is necessary for the existence of the tendon reflexes, were completely degenerated in the whole lumbar cord. Moreover, the columns of Clarke, and their ganglion cells in the grey substance, were affected with increasing intensity from the upper dorsal to the lumbar region.

Among the portions which have remained intact we must



quote the direct pyramidal tracts, and the "antero-external zones" of the posterior columns, and especially the marginal zone of Lissauer,† which has always been found hitherto much degenerated in ordinary tabes.‡ If this degeneration is constant in Duchenne's ataxy and is regularly wanting, as it seems to be up till now in Friedreich's ataxy, this will be a new anatomical character important to differentiate between these two diseases. (See Lissauer's diagram of the lumbar posterior cornu in *BRAIN*, October, 1888, p. 329.)

To sum up, after the results of the different autopsies to which we have just referred, we may conclude that from the anatomical point of view, Friedreich's disease is clearly distinguished from tabes and from the other combined scleroses.

1st. It is distinguished from tabes by the primary combined system, degeneration of the direct cerebellar, and of the crossed pyramidal tracts, by the pronounced atrophy of the cells and of the fine fibres of the columns of Clarke, whilst the marginal zone of Lissauer always remains intact. In tabes, this zone is, on the contrary, constantly degenerated, whilst the cells of the column of Clarke, the direct cerebellar, and the crossed pyramidal tracts are intact. In tabes frequent multiple degeneration of the peripheral nerves (motor and sensory nerve roots, Déjerine); in Friedreich's disease, exceptionally a few degenerated fibres in the nerve trunks (Rütimeyer). We do not, therefore, share on this point the opinion of Rütimeyer, who admits, as a symptom common to the two diseases, degenerations in different degrees of the peripheral nerves. There is certainly a considerable and characteristic difference in this respect between the two diseases. The only anatomical characters that they have in common would thus be the integrity of the grey matter of the anterior cornua, the sclerosis of the posterior

† H. LISSAUER, Beitrag zum Faserverlauf im Hinterhorn des menschlichen Rückenmarks und zum Verhalten desselben bei Tabes dorsalis. *Archiv für Psychiatrie*, Bd. XVII. p. 377.

‡ OPPENHEIM und SIEMERLING, Beiträge zur Pathologie der Tabes dorsalis und der peripherischen Nerven-Erkrankung. *Archiv f. Psych.*, Bd. XVIII., Heft. 1 and 2. W. B. HADDEN and C. S. SHERRINGTON. The Pathological Anatomy of a case of Locomotor Ataxy. *BRAIN*, Part XLIII., October, 1888, p. 329. "Throughout the lumbar region, Lissauer's tract is completely sclerosed."



columns (of Goll and of Burdach), especially in their middle part, with relative integrity of the antero-external bands and atrophy of the posterior roots.

2nd. Friedreich's disease is distinguished from the other combined sclerosis by the regularity of the seat of its system localisation, whilst in the other combined sclerosis we meet with the most varied lesions; and especially because the disease invades the structure of which is congenitally incomplete. In all the cases of Friedreich's disease which have been examined *post mortem*, the authors have been surprised by the smallness of the cord and of the bulbs which have always been found slender and atrophied, especially in their posterior part, as well as by the smallness of the nervous elements of these organs, cells and fibres, as seen by the microscope.

Friedreich's disease, which forms an absolutely distinct clinical group, presents, therefore, also anatomo-pathological characters, which allow us to class it apart in spinal diseases. It is a combined primary system sclerosis of the spinal cord, in which several systems of fibres have been affected from birth or during infancy, with an arrest of development (posterior columns, pyramidal, and direct cerebellar tracts), and degenerate before having attained their full growth.

#### PROGNOSIS AND TREATMENT.

Two things must be distinguished in prognosis; as regards the cause of the disease, and the gravity of the affection, *quoad vitam*. If Friedreich's disease does not directly threaten life, if the patients do not die from it, since those who are affected by it may reach a comparatively advanced age, thirty, forty years, and even more (when they are not cut off by an intercurrent affection, which is most usually the case), hereditary ataxy does not the less bear a grave prognosis, for it is progressive, in an extremely slow way, it is true, but without permitting us to hope for remissions in its fatal course. We know of no case of cure of this disease. It is true that hitherto it has only been diagnosed when it was incurable. Shall we be more

fortunate in the future, when we shall be able to recognise it from its first symptoms?

We have unfortunately but little to say about the treatment in face of such a prognosis. As prophylactic measures, in the case of predisposed children, we ought to advise good hygiene, hydrotherapy, rational gymnastics, careful education, and intelligent supervision, to prevent the child contracting fatal habits of masturbation, which would, doubtless, favour the evolution of the disease by exhausting an already feeble spinal cord. We have observed on several occasions excesses of onanism in the antecedents of those who suffered from this disease, as we have also pointed out alcoholism of the parents, of the father especially, in the family history.

As for treatment properly called, we may have recourse, without hoping too much from it to two different systems. First of all to electrotherapy. Application of continuous currents, stable and labile, to the vertebral column, by means of large electrodes (ten to fourteen milliamperes, three times a week). The patient generally feels well after the applications of electricity. He feels stronger, more active, less weak, and less uncertain on his legs. By continuing this treatment for several years, with regular intervals, we may, perhaps, obtain a decided delay in the progress of the disease. This is at least the impression which I have derived from the treatment, to which I have submitted the patient whose case has been related above. He has come to have the current applied for three years, omitting the applications two or three times each year, and he has told me that he found benefit from them. "I should not come so regularly," said he to me, "if I did not experience any benefit."

The second method is that of suspension, recommended by M. Charcot. They have obtained at the Salpêtrière some good results in this disease. I have suspended my patient more than a hundred times with a certain success. Speech has improved; the general condition has derived good from it. The patient has often found benefit from it, but the local symptoms of ataxy of the extremities and of weakness

of the legs have not been at all modified. The club-foot, the scoliosis, the nystagmus, the abolition of knee reflexes, the choreiform instability, all these exist after as before the treatment by suspension. Would more have been gained by employing Sayre's jacket, as M. Gilles de la Tourette proposed? We do not think so. But it is evident that we ought to try everything to combat a disease against which we do not really possess any efficacious means of treatment.

#### APPENDIX.

We must add to the cases which do not belong to Friedreich's disease that of J. Botkin.<sup>(63)</sup> By the courtesy of Prof. Dragomanoff, who wished me to translate it, I am able to give here a résumé of this case. Here is, first of all, the exact title of the article.

JAC. BOTKIN. Sloutchai Friedreich-oviboliezni. (A case of Friedreich's disease, *Medical Review*, Moscow, 1885, No. 1, p. 32-38).

Theodore Malych, age twenty-two, single, admitted November 7th, 1884, to the hospital of Yaroslaw. Mother hysterical; insane for two years after the birth of this son. Father died ten years ago, after having suffered for fifteen years from a convulsive affection, which probably had an analogy with the disease of his son, as all those who knew him affirm. The same mimicry, the same gestures, the same bearing, the same grinding of the teeth, the same movements of the eyes and of the head. The eldest brother of the patient had also in childhood convulsive attacks. His youngest brother has symptoms of "moral insanity." The patient began to walk only after his second year. He has often been ill, and in his illnesses frequently had convulsions. From eight to fifteen years of age pleurisy, scarlet fever, and typhoid fever. Each of these diseases accompanied by convulsions. Theodore Malych is timid, irascible, indolent, and obstinate. Expelled from the fifth class of the gymnasium on account of bad conduct. At sixteen years of age he enters at the special school as military surgeon's assistant. But headaches and diplopia compelled him to interrupt his

studies after seven months. After that swelling in the joints (rheumatism) for fourteen months. At the age of eighteen years (April-June, 1880), bad headaches, insomnia and melancholia. The latter lasted till December 6th. On this day on finding himself at the church of the convent of the Trinity he was taken with violent, irresistible laughter. The police took him to the hospital, where he remained four months. It is then that several symptoms of his real disease manifested themselves in the space of a few days, according to his own statement, with violent vomiting.

Present condition. Height, 167 cent.; weight, 137 Russian pounds. General pallor. Skull symmetrical. Sight normal on the right side. Left eye amaurotic. Normal reaction of pupils to light. Nystagmus. Sphincters normal. Copious salivation.

The bearing, the gestures, and the facial expression of the patient attract the attention especially. He stands erect, with the legs apart, the body thrown backwards, and leaning to the left. Head inclined backwards towards the right shoulder. Chin turned to the left and upwards, as in contracture of the sterno-mastoid. Hands crossed over the stomach. Hair thin. Apathetic expression. Jaws closed, and lips partially opened.

The voluntary movements are accompanied by a series of involuntary movements. The head is agitated by zig-zag movements, the teeth are ground. The eyes go from left to right. The arms are elevated and depressed, are flexed and extended. The hands execute movements of prehension. The body undergoes convulsive movements, forward, backward, to the left, to the right, is bent and again straightened. The feet are separated widely during walking. Trembling of the lower extremities in consequence of involuntary muscular contractions. In walking the patient sways; he is thrown from side to side. The direction of locomotion is always in a zig-zag. The legs give way sometimes and the patient falls.

The ataxy is so marked in the upper extremities that the patient has difficulty in dressing himself, and buttoning his clothes. He writes badly, and he cannot thread a needle.

When he closes his eyes, there is no increase of the ataxy, either static or dynamic. Romberg's symptom not present.

The more complicated the movement is, the more attention it demands, and the more agitated the patient is, the more do the involuntary contractions increase. The ataxy is especially pronounced when the patient walks or when he endeavours to narrate his history in a clear and distinct manner, for he never succeeds in it. It is impossible to describe the complication of the involuntary movements which are then observed. One must see them to understand to what extent they are developed.

At first sight, these movements appear disorderly, but after an attentive examination it is noticed: 1st. To each voluntary movement a definite group of involuntary movements corresponds; 2nd however numerous and varied they may be, these movements cease rapidly immediately the patient lies down horizontally and ceases efforts. The grinding of the teeth and the ocular movements form the only exception and cease only during sleep, when all the other involuntary movements also cease. Clonic contraction of the lower extremities as soon as the toes rest on the ground or on the mattress. Spasms of the muscles of the neck and of the arms. These spasms increase with the voluntary movements, and render the latter difficult, as also passive movements.

Speech is varied in tone. Far from being monotonous the sound of the voice is changed during conversation or reading; the patient passes involuntarily from one tone to another. He pronounces certain words indistinctly, does not pronounce always the end of the words. The remission of symptoms is a peculiarity of his disease. Thus sometimes he speaks much more distinctly.

Muscular enfeeblement. Dynamometer, 40 in the right, 25 in the left. The patient when lying down cannot lift his leg. If it is raised for him, he allows it to fall again.

*Reflexes*; plantar and cremasteric, distinct, normal; abdominal, gluteal, epigastric, scapular, but slightly marked.

Patellar reflexes and Achilles tendon reflexes *very exaggerated*. No ankle clonus.

*Sensation.* Rheumatic-like pains in the extremities. Vertigo. The patient localises exactly the lightest touch and the position of his limbs in space.

Spinal irritability. Slight hypho-scoliosis. Electric reactions normal.

*Psychical manifestations.* The patient is habitually apathetic, often excitable, and ready to laugh causelessly. Memory good. Imagination unimpaired.

In the discussion following the case, M. Botkin maintains that the presence of pareses, of contractures, and of exaggerated patellar reflexes does not exclude Friedreich's disease. He adds, however, that there exist in his patient two symptoms which have not been hitherto observed in this disease, unilateral amaurosis and grinding of the teeth. In any case, says he, even when these two symptoms would indicate a focal lesion, they cannot be regarded as due to a multilocular sclerosis. If we admit, with Féré and Erb, that Friedreich's disease is an intermediate form between disseminated sclerosis and tabes, there is nothing astonishing, concludes Botkin, in the fact that the symptoms of both lesions may be met with in this intermediate form, as has occurred in my case.

It will be sufficient to refer to the comparative table that we have given above of the differential diagnosis between Friedreich's disease and disseminated sclerosis to be convinced that Dr. Botkin's case does not belong at all to Friedreich's disease. We think that similar cases will not henceforth be accepted under the heading of this disease.

With regard to my provisional clinical classification of the combined scleroses, M. Dejerine writes to me :—

“Your type No. 1 (ataxic paraplegia of Gowers), and your type No. 3 (ataxospastic tabes of Grasset) are only one and the same type, clinically and anatomically, as I have established in 1884 and 1886. It is nothing else than *ataxo-paraplegic tabes of Dejerine*. I have even, unless I am mistaken, shewn you the preparations of it.”

Not only has M. Dejerine shewn me numerous preparations of combined scleroses, but he has admitted me with the greatest courtesy to his admirable practice at the

Bicêtre, where he has given me permission to examine several of his patients affected with combined sclerosis. I am happy to express here my sincere gratitude to him. His words have been fully opened to me, as also those of Professor Charcot at the Salpêtrière, for the observations I was desirous of making on Friedreich's disease and the combined sclerosis. I owe thanks also to Professor Prevost, of Geneva, who has had the kindness to show me the preparations of the cord of the patient whose case he has published, with the autopsy, in the Archives de Physiologie, 1877, and Professor Schiff, who has shewn me the cord sections which he possesses, belonging to the first patient examined *post-mortem* by Friedreich.

I must state here that I have not touched upon the study of the combined sclerosis, and that the historical investigation, in particular, of this disease remains quite untouched, and has nothing to do with my clinical classification. I have not occupied myself at all in this work of the examination of the history of the combined sclerosis, and I had no occasion to occupy myself with it. It would have been a digression. I have mentioned the combined sclerosis only incidentally in the chapter on diagnosis, and with the sole object of showing that they could not be confounded with Friedreich's disease, when the latter presents itself under one of the forms corresponding with the four types which I have described. Now, from this point of view, it was necessary for me to separate into two distinct clinical types, ataxic paraplegia properly called and spastic ataxy. My classification has no other significance, and especially would not attempt to trench upon a historical question. If I had had to treat of this question, I venture to say that I should have recognised openly the legitimate place, and the importance which ought to be attributed, in a historical study of the combined sclerosis, first of all to the case recorded by M. Prevost which dates from 1877, afterwards to the works of Westphal, Kahler and Pick, Babesiu, Raymond, Damaschino, Hopkins, Ballet and Minor, &c.; finally, especially to the memoir published in 1884 by M. Dejerine, which marks a decisive epoch in this study, and where is found for the first time, unless I am



mistaken, the complete picture of ataxo-paraplegic tabes, two years in fact before the works of Gowers and Grasset, which were printed in 1886, which is seen clearly from the bibliographical notices which I have given.

In conclusion, I shall draw attention to the work of Dr. E. Huet, *on chronic chorea*, Paris, 1888-1889, where will be found (p. 190) an interesting page devoted to the differential diagnosis of Friedreich's disease and chronic chorea, both of which are family and hereditary affections, with motor inco-ordination.

But whilst in Friedreich's disease there is choreiform instability during rest, in hereditary chorea we observe small separate, multiplied, varied and irregular movements. Romberg's sign is not present in the latter disease, nor yet nystagmus. The affections of speech differ greatly in the two diseases. The knee jerks are rather exaggerated in chorea, which is especially an affection of adult age, whilst Friedreich's disease appears, as we know in childhood and youth.

#### BIBLIOGRAPHY.

(<sup>1</sup>) N. FRIEDREICH, A paper read before the Congress of German naturalists and physicians at Speyer (Sept. 18th, 1861), published in the "*Beilage zum Tagblatt der 36. Versammlung deutscher Naturforscher und Aerzte in Speyer*," p. 10, &c.

(<sup>2</sup>) MARIUS CARRE, De l'ataxie locomotrice progressive. *Thèse de Paris*, No. 131, Aug. 13, 1862, p. 35. obs. I., pp. 61 and 81.

(<sup>3</sup>) N. FRIEDREICH, Ueber degenerative Atrophie der spinalen Hinterstränge. *Virchow's Archiv*, vol. xxvi, 1863, pp. 391 and 433, vol. xxvii, 1863, p. 1. Translated in *Archiv. gén. de méd.*, t. II., Dec., 1863 and t. III., March, 1864 (6th series).

(<sup>4</sup>) PAUL TOPINARD, De l'ataxie locomotrice. Paris 1864 (obs. CCXV, p. 384). See also pages 141, 156, 169, 194 to 198, 211, 268, 326, 361, 369, 384 to 387.

(<sup>5</sup>) M. CARRE, Nouvelles recherches sur l'ataxie. Paris, 1865.

(<sup>6</sup>) ALFRED CARPENTER, M.D. (of Croydon). "Two cases of muscular Anæsthesia." Read before the Medical Society of London (Nov. 13, 1871). *Lancet*, Dec. 2, 1871, vol. ii., p. 779, and *Med. Times and Gaz.*, Sept. 7, 1872, vol. ii., p. 264.

(<sup>7</sup>) BRADEURY, *Brit. Med. Journal*, 1871, p. 565 (Judson Bury), Locomotor Ataxy in a young man of 18 years after excessive onanism, *Brit. Med. Journ.*, 1871, vol. ii., p. 499 (Soca).



(<sup>8</sup>) KELLOGG, Two cases of Locomotor Ataxy in Children. *Archiv of Electrol. and Neurol.*, New York, vol. ii., p. 182, 1875.

(<sup>9</sup>) N. FRIEDREICH, Ueber hereditäre Ataxie. Wanderversammlung südwestdeutschen Irrenärzte zu Heppenheim. *Allgemeine Zeitschrift für Psychiatrie*, vol. xxxii., Berlin, 1875, p. 539.

(<sup>10</sup>) N. FRIEDREICH, Ueber statische Ataxie und ataktischen Nystagmus. *Archiv. für Psychiatrie*, 1876, VII., p. 235-238.

(<sup>11</sup>) N. FRIEDREICH, Ueber Ataxie mit besonderer Berücksichtigung der hereditären Formen. *Vichow's Archiv*, Bd. 68, 1876, p. 145, and Bd. 70, 1877, p. 140.

(<sup>12</sup>) DRESCHFELD, Family Predisposition in Locomotor Ataxy. *Manchester and Liverpool Hospital Reports*, vol. ii., 1876, p. 93.

(<sup>13</sup>) SHAW, J.-C., "A case of Ataxia in a Child." *Trans. Americ. Neurol. Association*, New York, 1877, ii., pp. 85 and 87.

(<sup>14</sup>) LEYDEN, Klinik der Rückenmarkskrankheiten, t. II, 1875, pp. 325, 335 and 370; French trans. by E. RICHARD and C. VIRY, Paris, 1879, pp. 596, 629.

(<sup>15</sup>) KAHLER UND PICK, Ueber combinirte Systemerkrankungen des Rückenmarks. *Archiv. f. Psych. und Nervenkrankheiten*, v. Westphal., Bd. VIII., 1878, p. 251.

(<sup>16</sup>) ERB, Krankheiten des Rückenmarks. Handbuch der speciellen Pathologie und Therapie von Ziemssen, Bd. XI, 1878, 2 Hälfte, 2 Abtheilung. second ed., p. 601.

(<sup>17</sup>) EULENBURG, Lehrbuch der Nervenkrankheiten, Bd. II., 1878, p. 458.

(<sup>18</sup>) VULPIAN, Maladies du système nerveux, t. I., p. 245, 1879, and t. II., 1886, p. 226 (Scléroses primitives combinées) and p. 239 (Maladie de Friedreich)

(<sup>19</sup>) MÖBIUS, Ueber die hereditären Nervenkrankheiten. *Sammlung klinischer Vorträge von Volkmann*, No. 171, 1879.

(<sup>20</sup>) F. SCHULTZE, Ueber combinirte Strangdegenerationen in der Medulla spinalis. *Vichow's Archiv*, Bd. 79, p. 132, 1880.

(<sup>21</sup>) A. SEELIGMÜLLER, Sclerose der Hinterstränge. Handbuch der Kinderkrankheiten v. Gerhardt, 1880, p. 187.

(<sup>22</sup>) HENOCHE, Ataktische Symptome durch Genitalreizung bei einem siebenjährigen Knaben. *Wiener med. Bl.* 1880, No. 12.

(<sup>23</sup>) A. SEELIGMÜLLER, Hereditäre Ataxie mit Nystagmus. *Archiv. f. Psychiatrie und Nervenkrankheiten*, Bd. X., p. 222, 1880.

(<sup>24</sup>) SCHMID (in Alstätten), Ueber hereditäre Ataxie. *Correspondenz-Blatt für Schweizer Aerzte*, February 15, 1880, p. 97.

(<sup>25</sup>) GOWERS, A Family affected with Locomotor Ataxia. *Trans. of the Clin. Society*, London, vol. xiv., p. 1, 1880. Reports of Meetings of Oct. 8, 1880, in *The Lancet*, Oct. 16, 1880, t. II., p. 618; *British Med. Journal*, Oct. 16, 1880; *The Medical Times and Gazette*, vol. ii., p. 478, 1880; *Berliner klinische Wochenschrift*, Nov. 15, 1880, No. 46, p. 660; *Schmidt's Jahrbücher*, 1882, t. 196, p. 93 (abst. by Möbins).

(<sup>26</sup>) HOLLIS (W. A.) Locomotor Ataxy in a Boy. *Brit. Med. Journal*, 1880, vol. ii., p. 167 (read at the East Sussex District meeting).

(<sup>27</sup>) GRASSET, Traité pratique des maladies du système nerveux. Second ed., Paris, 1881, p. 321.

(<sup>28</sup>) W. A. HAMMOND, On the so-called Family or Hereditary Form of Locomotor Ataxy. *Journal of Nerv. and Mental Disease*, 1882, p. 484 (read before the American Neurological Association, June 23, 1882). See *Archives de Neurologie*, 1883, p. 117, No. 13, *Neurologisches Centralblatt*, 1882, Dec. 15, p. 557 and *Schmidt's Jahrbücher*, 1884, t. 204, p. 26.

(<sup>49</sup>) AURELIO BIANCHI, La malattia del Friedreich. *Gazzetta degli Ospitali*, No. 99, Dec. 10, 1882, p. 785.

(<sup>50</sup>) SCHULZ, Richard, Ataxia im Kindesalter. *Centralblatt für Nervenheilkunde*, von Erlenmeyer, Coblenz, 1882, p. No. 4.

(<sup>51</sup>) P. DAVIDSON, M.B., Two Cases of Ataxia in Children. *Med. Times and Gazette*, London, 1882, vol. i., p. 410. — a) OXLEY, Ataxic Condition accompanying Emaciation, — b) POLLARD, Ataxic Condition following Rheumatic Fever.

(<sup>52</sup>) D'ARCY POWER, A Case of Hereditary Locomotor Ataxy. *St. Bartholomew's Hosp. Reports*, 1882, XVIII., p. 305-308.

(<sup>53</sup>) FÉRÉ, Ataxie héréditaire. Maladie de Friedreich. Sclérose diffuse de la moelle et du bulbe. *Progrès médical*, X, No. 45, p. 890, Nov. 11, 1882.

(<sup>54</sup>) AUG. BROUSSE, De l'ataxie héréditaire (Maladie de Friedreich), Paris, 1882.

(<sup>55</sup>) G. LEUBUSCHER, Ein Fall von Tabes dorsalis im frühesten Kindesalter. *Berliner klinische Wochenschrift*, Sept. 25, 1882, No. 39, p. 590.

(<sup>56</sup>) F. W., Diffuse Sclerosis of the Spinal Cord and Medulla Oblongata. Disease of Friedreich, Leading Article in the *Philadelphia Medical Times*, 1882-1883, t. XIII., Feb. 24, 1883, p. 372.

(<sup>57</sup>) SCHULZ, Richard, Nachtrag zur Ataxie im Kindesalter. *Centralblatt für Nervenheilkunde, Psychiatrie und gerichtlichen Psychopathologie* von Erlenmeyer, vol. vi., No. 12, June 15, p. 266.

(<sup>58</sup>) SEPPILLI, Atassia ereditaria o malattia di Friedreich. Rassegna critica del. dott. Giuseppe, S. *Rivista sperimentale di Frenatria e di medicina legale*, Anno IX., 1883, p. 335.

(<sup>59</sup>) Leading article on Friedreich's disease. *Brit. Med. Journal*, Mar. 31 1883, vol. i., p. 627 and 628.

(<sup>60</sup>) A. ERLENMEYER, Zur Lehre von den Coordinationsstörungen im Kindesalter. *Centralblatt für Nervenheilkunde*, Sept. 1, 1883, No. 17, p. 385.

(<sup>61</sup>) L. RÜTIMEYER, Ueber hereditäre Ataxie. *Virchow's Archiv*, Bd. 91, pp. 106, 115, 1883.

(<sup>62</sup>) F. SCHULTZE, Die Pathologie und pathologische Anatomie der hereditären Ataxie. *Neurologisches Centralblatt*, 1883, No. 13, p. 290. (Die acht Wanderversammlung der südwestdeutschen Neurologen und Irrenärzte. Bericht von Dr. Laquer).

(<sup>63</sup>) H. WÆLLE (in Wattwyl), Zwei neue Fälle von hereditären Ataxie. *Correspondenzblatt für Schweizer Aerzte*, No. 2, Jan. 15, 1884, p. 33.

(<sup>64</sup>) J. TEISSIER, Maladie de Friedreich. Ataxie héréditaire avec dégénérescence des faisceaux postérieurs de la moelle. *Lyon Médical*, 16th year, t. XLVI., p. 45, No. 19, May 11, 1884.

(<sup>65</sup>) J. M. CHARCOT, L'ataxie héréditaire. *Gazette des Hôpitaux*, April 29, 1884, No. 50, p. 303.—(*Id.*) *Progrès médical*, April 29, 1884.

(<sup>66</sup>) J. M. CHARCOT, same Lecture reported by Dr. MILLIOTTI in *Gazzetta degli ospitali*, No. 67 and 68, Aug. 1884.

(<sup>67</sup>) JACCOUD, Traité de Pathologie, seventh edit., 1883.

(<sup>68</sup>) MASSALUNGO, Roberto, Della malattia di Friedreich. *Rivista Veneta di Scienze mediche*, Nov., 1884, Anno. I., tomo I., p. 393, fascicolo V. (with plate representing the brother and sister standing, legs apart, eyes fixed to the ground).

(<sup>69</sup>) G. MUSSO, Sulla Malattia del Friedreich. Atassia locomotrice ereditaria. *La Rivista clinica di Bologna*. October, 1884.

(<sup>30</sup>) R. LONGUET, La maladie de Friedreich. Ataxie héréditaire. *Union Médicale*, vol. i., May 18, 1884, p. 877 (critical digest based upon fifty cases).

(<sup>31</sup>) J. A. ORMEROD, On the so-called Hereditary Ataxia first described by Friedreich, a critical digest. *Brain*, vol. VII., p. 105, April, 1884 (list of forty cases).

(<sup>32</sup>) E. S. SEGUIN, Clinical Report of two sets of cases of Friedreich's Disease. Hereditary or Family Ataxy. *The Medical Record*, New York, June 13, 1885, No. 24, XXVII., p. 645-647.

(<sup>33</sup>) SINKLER, Two cases of Friedreich's Disease. *Medical News*, 1885, July 4. American Neurol. Association, Transactions of Eleventh Annual Meeting, June 17, 1885 (*Journal of Nervous and Mental Disease*, July, 1885).

(<sup>34</sup>) J. A. ORMEROD, An Account of Two Ataxic Families. *Med. Chir. Transact.*, vol. lxxviii., p. 147, 1885.—See also *The Brit. Med. Journal*, Feb. 28, 1885, p. 435. Report of Meeting of Royal Med. and Chir. Soc., Feb. 24, 1885. On Hereditary Locomotor Ataxy, quoted in *Neurologisches Centralblatt*, No. 16, p. 382, August 15, 1885.

(<sup>35</sup>) PALMA, Malattia del Friedreich. Relazione sulla cura delle acque del Gurgitello praticata in Napoli, 1885.

(<sup>36</sup>) F. FAZIO, Un altro caso della cosiddetta atassia ereditaria o tabe del Friedreich. *Rivista clinica e terapeutica*, p. 73, 1885 (Napoli), Anno VII.

(<sup>37</sup>) BUZZARD, Discussion on Dr. Ormerod's paper before the Royal Med. and Chir. Society, Feb. 24, 1885. *Brit. Med. Journal*, Feb. 28, 1885, p. 435, and *The Lancet*, Feb. 28, 1885, t. I., p. 383 (*Neurolog. Centralblatt*, No. 16, August 15, 1885, p. 383. Rendiconto della R., academia Med. Chir. di Londra, *Riforma medica*, March, 1885).

(<sup>38</sup>) K. FOWLER, *ibid.*

(<sup>39</sup>) RAFFAELE VIZIOLI, La malattia di Friedreich (atassia ereditaria), *Giornale di Neuropatologia*, 1885, fasc. 1 and 2, p. 7-41, fasc. 3 and 4, p. 145-177.

(<sup>40</sup>) W. EVERETT SMITH, Hereditary or Degenerative Ataxia. Six cases in one family. Death of one case and autopsy. *Boston Medical and Surgical Journal*, Oct. 15, 1885, vol. cxiii., p. 361 (with photographs and drawings of sections of the cord. See *Neurolog. Centralbl.*, 1885, p. 536).

(<sup>41</sup>) MORTON PRINCE, A Case of Spinal Ataxia without Loss of Sensation and with increased Patellar-Tendon Reflex. A Contribution to the Study of Spinal Ataxy. *Boston Med. and Surg. Journal*, vol. cxiii., 1885, p. 371.

(<sup>42</sup>) F. RAYMOND, Tabes dorsalis et Tabes spasmodique. Dictionnaire encyclopédique des sciences médicales de Dechambre, Paris, 1885, t. XV, 1st part, p. 288, and t. XV. 2nd part, p. 417.

(<sup>43</sup>) J. BOTKIN, Seluchai Fridriecho voi bolczni. *Med. Oboz.*, Mosk., 1885, XXIII, p. 32-38.

(<sup>44</sup>) MAC ALISTER, Friedreich's Ataxy. *Brit. Med. Journal*, January 2nd, 1886, p. 19.

(<sup>45</sup>) GALASSI, Malattia di Friedreich. *Bolletino della Societa Lancisiana degli ospitali di roma*, 1886.

(<sup>46</sup>) J. DÉJÉRINE, L'hérédité dans les maladies du système nerveux. Paris, 1886, p. 195. Ataxie héréditaire (maladie de Friedreich).

(<sup>47</sup>) GILLES DE LA TOURETTE, Etudes cliniques et physiologiques sur la marche, p. 50. *Thèses de Paris*, 1886.

(<sup>48</sup>) DESCROIZILLES, Un cas d'ataxie héréditaire. *Progrès médical*, No. 28, July 10, 1886, p. 569.

(<sup>49</sup>) JUDSON S. BURY, A Contribution to the Symptomatology of Friedreich's disease. Reprinted from *BRAIN*, part xxxiv, July, 1886, 9th year, p. 145.

(<sup>69</sup>) O. VIERORDT, Beitrag zur Kenntniss der Ataxie. *Berliner klinische Wochenschrift*, May 24, 1886, No. 21, 333.

(<sup>71</sup>) GLYN, Case of Friedreich's Disease. *Liverpool Med. and Surg. Journal* 1887, p. 191.

(<sup>72</sup>) FERRIER, Case of Friedreich's Disease. *Brit. Med. Journal*, June 4, 1887, vol. i., p. 1214.

(<sup>73</sup>) R. STINTZING, Ueber hereditäre Ataxie. *Münchener medicinische Wochenschrift*, No. 21, 1887 (Separatabdruck).

(<sup>74</sup>) G. NEWTON PITT, On a Case of Friedreich's Disease. Its clinical history and *post-mortem* appearances. *Guy's Hospital Reports*, vol. xliv. (1886-1887) London, 1887, p. 369 (with ten illustrations of spinal sections).

(<sup>75</sup>) CHARCOT, La maladie de Friedreich. Report of Lecture in *Progrès médical*, No. 23, June 4, 1887, p. 453, 15th year.

(<sup>76</sup>) CHARCOT, La maladie de Friedreich. Diagnostic différentiel d'avec l'ataxie locomotrice et la sclérose en plaques. *Gazette des Hôpitaux*, No. 52, April, 1887, p. 413.

(<sup>77</sup>) L. RÜTIMEYER, Ueber hereditäre Ataxie. Ein Beitrag zu den primären combinirten Systemerkrankungen des Rückenmarkes. *Virchow's Archiv*, Band 110, Nov. 1, 1887, p. 215.

(<sup>78</sup>) MORITZ FREYER, Zur Tabes im jugendlichen Alter. *Berliner klinische Wochenschrift*, February 7, 1887, No. 6, p. 91.

(<sup>79</sup>) M. MASTIN, Two Generations of Hereditary or Congenital Ataxia, *Med. News*, vol. li., No 9.

(<sup>80</sup>) SOARES DE SOUZA, Estudio clinico de ataxia hereditaria de Friedreich. *Thèse de Rio de Janeiro*, 1887.

(<sup>81</sup>) F. MENDEL in Essen a. d. Ruhr, Zur Lehre von der Ataxie. Friedreich'sche Tabes. *Berliner klin. Wochenschrift*, Oct. 10, 1887, No. 41, p. 771.

(<sup>82</sup>) P. BLOQ, Un cas de maladie de Friedreich. *Archives de Neurologie*, March, 1887, p. 217, vol. xiii., No. 38.

(<sup>83</sup>) ALBERT ADAMKIEWICZ, Die degenerativen Krankheiten des Rückenmarks. Anatomisch und klinisch bearbeitet. Stuttgart, 1888, p. 101. Hereditäre Form der Ataxie.

(<sup>84</sup>) Dr. C. L. DANA, Friedreich's Disease or Hereditary Degenerative Ataxia. *The Medical Record of New York*, October 1, 1887, p. 465.

(<sup>85</sup>) AD. SEELIGMÜLLER, Lehrbuch der Krankheiten des Rückenmarks und Gehirns, &c. (Wreden's Sammlung kurzer med. Jahrbücher Bd. xii.), 1887. Hereditäre Ataxie, p. 194 et 201.

(<sup>86</sup>) JOFFROY, Sur la maladie de Friedreich. *Bulletin medical*, No. 16, February 26, 1888, p. 247.

(<sup>87</sup>) CHARCOT, Leçons du mardi à la Salpêtrière, March 13, 1888 (14th lecture), p. 253, and April 10, 1888, p. 326 (17th lecture).

(<sup>88</sup>) GEORGE B. SHATTUCK, Three Cases of Hereditary Locomotor Ataxia (Friedreich's disease). *Boston Medical and Surgical Journal*, February 16, 1888, vol. cxvii., No. 7, p. 168, and p. 175.

(<sup>89</sup>) GILLES DE LA TOURETTE, BLOQ, HUET, Cinq cas de maladie de Friedreich. Nouvelle iconographie de la Salpêtrière, No. 2, March and April, 1888, p. 45 ; No. 3, May and June, 1888, p. 114.

(<sup>90</sup>) WELLS, Friedreich's disease—a group of five cases. *Journal of American Med. Association*, 1888.

(<sup>91</sup>) F. VINCENT SOCA (de Montevideo), Un nouveau cas de maladie de Friedreich. Nouvelle iconographie de la Salpêtrière, No. 4, July and August, 1888, p. 155 ; No. 5, September and October, 1888, p. 183-190.

(<sup>92</sup>) FRANCOIS VINCENT SOCA, Etude clinique de la maladie de Friedreich. *Thèse de Paris*, No. 17, November 14, 1888.

(<sup>93</sup>) C. BERNARDEI, Sulla malattia del Friedreich. Clinica medica generale della R. Università di Pisa. Estratto del giornale *La Riforma medica*. Anno iv., maggio, 1888.

(<sup>94</sup>) ORMEROD, Critical digest. On the Morbid Anatomy of Friedreich's Disease. *BRAIN*, October, 1888, p. 406.

(<sup>95</sup>) J. MICHEL CLARKE, An Account of Three Cases of Friedreich's Disease or Hereditary Ataxia. *The Lancet*, March 23, 1889, p. 570.

(<sup>96</sup>) DR. SUCKLING, Hereditary Ataxia, or Friedreich's Disease (three cases). *British Medical Journal*, No. 1481, May 18, 1889, p. 1119.

(<sup>97</sup>) ORMEROD, J. A., Some Further Observations on Friedreich's Disease. *BRAIN*, vol. x., parts xxxix. and xl., January, 1888, p. 461.

(<sup>98</sup>) ERLICKI et RYBALKIN, Zur Frage über die combinirten Systemerkrankungen des Rückenmarkes. *Archiv für Psychiatrie*, Bd. xvii., 1886, p. 693.—Combined sclérosis of posterior fasciculi and of crossed pyramidal tracts, with complete integrity of direct cerebellar fasciculi. Ataxo-paraplegic symptoms. Interesting discussion; the authors wrongly consider the case as one of Friedreich's disease.

(<sup>99</sup>) L. RÜTIMEYER, Ueber die anatomische Localisation der hereditären Ataxie. (*Correspondenz-Blatt für Schweizer Aerzte*, 1888, No. 8, April 15, p. 252.)

(<sup>100</sup>) C. PAPENHAUSEN, Ueber Friedreich'sche Krankheit. Thesis, Berlin, 1888.

(<sup>101</sup>) J. F. C. GRIFFITH, A Contribution to the Study of Friedreich's Ataxia. *Americ. Journal of the Med. Sc.*, October, 1888.

(<sup>102</sup>) E. W. SMITH, Postero-lateral Spinal Sclerosis (generic origin), or Generic Ataxia. *Boston Journal*, March 1, 1888.

(<sup>103</sup>) ALIX JOFFROY, Observation de la maladie de Friedreich. *Gazette hebdomadaire*, March 9, 1888, No. 10, p. 149.—Non-degenerative atrophy of muscles of right shoulder. Facial paresis, possibly of bulbar origin. Temporary diplopia. Tubercular family.

(<sup>104</sup>) CHARLES L. DANA, A case of ataxic paraplegia, with autopsy. *BRAIN*, part xliv., January, 1889, p. 490.

(<sup>105</sup>) H. SURMONT, Note sur une névropathie héréditaire (maladie de Friedreich à marche retardée). *Bulletin médical du Nord*, Lille, 1889.

## Reviews and Notices of Books.

- I.—*The Anatomy of the Central Nervous Organs in Health and in Disease.* By DR. HEINRICH OBERSTEINER. Translated with Annotations and Additions by ALEX. HILL, M.A., M.D., M.R.C.S. London: Charles Griffin & Co., 1890.
- II.—*Twelve Lectures on the Structure of the Central Nervous System.* By DR. LUDWIG EDINGER. Second Revised Edition, with 133 Illustrations, Translated by WILKS HALL VITTM, M.D. Edited by C. EUGENE RIGGS, A.M., M.D. Philadelphia and London: F. A. Davis, 1890.

### I.

THE original of this work was reviewed in the number of this journal for April, 1888, and the hope was then expressed that it would be translated into English. This has now been done by Dr. Alexander Hill, who has also made various annotations and additions to the original work. The translator has also added a chapter in the appendix on his theory of the rotation of the great brain—a full account of which was published in 1885 at Cambridge under the title of “The Plan of the Central Nervous System,” and also a valuable glossarial index, besides inserting several fresh figures. Further, Professor Obersteiner has added much new matter and made numerous alterations so that, as the translator states in his preface, the present volume may fully claim to have the value of a new edition.

As the different sections of the work were described in detail in the review of the German edition it will here suffice to discuss the various annotations which have been made by the translator, and especially the appendix and the index for which he is responsible.

In the first section on methods, the process, first described by Barrett, of imbedding in celloidin in the usual way and afterwards taking out the spirit by water and then cutting the mass in the frozen state is recommended. In Section II. on Morphology,

Professor Hill's recent researches on the development of the nervous system from the epiblast, and the division of the cells into nemoblasts and spongio-blasts (supporting cells) is detailed.

In Section III. on the Nervous Constituents, the translator's theory with regard to "granules" is given, and a parallel is drawn between the granules of the "inner nuclear layer" of the retina and those of the olfactory bulb, and he looks upon them as the nucleus of a bi-polar cell with non-medullated processes.

In Section IV., the Histology of the Spinal Cord, is enriched by a short account of Gaskell's well-known researches on the Sympathetic System; and in the same section the question as to whether the posterior sensory roots of the cord are connected with cells or with a plexus is discussed, and here Dr. Hill takes the latter view, while the probability of the former being correct is maintained by the author. Further on, Sherrington's researches on counting the number of degenerated fibres and recording their position in the cord after cortical destruction, are mentioned. In the description of the corpus callosum on p. 347, it is stated by Professor Obersteiner that its fibres pass into the external capsule: this assertion the writer has not been able to confirm in the quadrumana, as here the fibres of the corpus callosum pass across those of the external capsule on their way to the operculum.

Besides the additions above noted, the English edition contains in the appendix a chapter by the translator on his theory of the rotation of the Great Brain, which he had previously propounded in his thesis on the Plan of the Central Nervous System in 1885. In this rotation the author considers that a loop or rink is formed, bringing the part which was at first in front on to the under surface of the back, so that the foramen of Monro, which in the embryo opens into the posterior part of its ventricle, in the adult opens into the front of the ventricle; he further states that this alteration in the position of the Great Brain was the almost necessary deduction from certain conclusions as to the connections of the olfactory tract with the anterior end of the optic thalamus, viâ the fimbria, fornix, descending pillar of the fornix, corpus mamillare and bundle of *Vicq d' Azyr*. As evidences of this connection, it is stated that in the hardened brain of the ox, the fornix, pyriform lobe, and olfactory bulb can be broken off in one piece, and that the fascia dentata is probably a continuation backwards of the glomerular layer of the olfactory bulb. As this connection is held as strong evidence to prove the rotation, it would be just advisable to consider whether it really



exists. The mere fact of hardened portions of the brain breaking off in one piece can hardly be taken as proof of functional connection, and as far as the writer has been able to ascertain microscopically, no definite connection can be made out—at any rate in the *quadrumanus*—that the olfactory tract is in connection with the fascia dentata. It can be seen in microscopical sagittal sections that although the pyriform lobule is situated immediately in front of the cornu ammonis and the fascia dentata, there is no connection between them; they are separated by the anterior end of the descending cornu of the lateral ventricle and by the fibres of the fimbria or alvens, and no fibres can be made out connected with the two bodies; and the same thing can be made out in the brains of *rodentia* and *carnivora*. Until, therefore, further evidence is forthcoming it can hardly be laid down that the olfactory tract passes into the fascia dentata. The pyriform lobule has sometimes been described as part of the uncinatæ convolutions, no doubt because the cortex on the surface is more or less continuous—though on page 276 of this work it is stated that in many animals the lobule is separated from the rest of the hemisphere by the scissura limbica—but on cutting through this cortex the separation can readily be seen.

With regard to the rotation of the brain, it is assumed that the anterior end of the temporal lobe is fixed to the ethmoid bone by the olfactory bulb and tract, and that the rest of the brain rotates forwards on its transverse axes. It is difficult to understand how this slender nerve can fix the temporal lobe so as to allow sufficient traction to be exerted to produce the fan-like arrangement of the fissures in the Island of Reil, as stated in the text. Also if the temporal lobe is the fixed point, it is difficult to explain the length of the olfactory tracts and the distance of the apex of the temporal lobe from the ethmoid bone in *carnivora* and *rodentia*, for the fixation must be assumed to take place in all the *mammalia*. One would like to know whether the appearances cannot be explained by the flexures which take place in the growth of the brain without calling in the aid of a theory of rotation. In the lowest *mammalia*, such as the kangaroo, the frontal region is slightly developed and the temporal region is very far back, whilst the cornu ammonis and fascia dentata extend so far forward as almost to take the place of the gyrus fornicatus; but as we ascend the animal scale the frontal region is pushed forwards and the temporal region downwards and forwards, so that in the *quadrumanus* and man owing to the high development of the so-called motor region, the tip of the temporal lobe comes



very near to the ethmoid, and the cornu ammonis does not make its appearance till the hinder end of the splenium is reached.

There is one addition to the English edition, for which our special thanks are due to Dr. Hill, and that is the Glossarial Index at the end of the book. He has taken great trouble to give an index of the German names used in the translation with the Latin and English names, and in many cases the French equivalents.

It would not be out of place here to raise a protest against the employment of a different word in every different language for the various parts of the brain, and we would urge that all neurologists should use the language common to all, viz.:—Latin. For instance, the claustrum is called *Vormauer* in German and *avant-mur* in French; and when we come to the *Hirnschenkel-schlinge* it takes the student of other nations a great deal of trouble to find out that it is the *ansa peduncularis*. Other words as the *äusserer Kniehöcker* (*corpus geniculatum externum*), *Strickkörper* (*corpus restiforme*) require the student to have a medical dictionary. In these days of international reading surely the Latin equivalent would save a great deal of unnecessary trouble, which Dr. Hill has here made an important step in alleviating. It is some satisfaction to feel that English neurologists have not erred in a similar manner, or we might have such words as the “front-wall,” the “brain-shank-loop,” the “outer knee-hump,” and the “rope-body,” as equivalents for the above words.

We must congratulate Dr. Hill on his excellent translation,—as it makes all the difference in the world to the reader whether a good English style is pursued or only a mere translation is offered, in which the foreign construction can be everywhere detected—and on the completion of a task which will bring Professor Obersteiner's valuable work within the reach of all English students of neurology.

## II.

A REVIEW by Dr. James Anderson, of the first German Edition of Dr. Edinger's Lectures has already appeared in *Brain* (Jan. 1886). A second edition was published in 1889, which has now been translated into English, and edited by the above-named authors.

Dr. Edinger states in the preface to his second edition that it

has undergone many changes, and has been added to in some respects. The chapters on histology and histogenesis have been entirely re-written, as also the sections on the oculo-motor, the acoustic, and the fibres of the deep marrow. The recent discoveries on the course of the tracts in the posterior tracts has had the effect of simplifying the description of that part. Dr. Edinger further states that he has embodied a treatise of the comparative anatomy of the nervous system based on his personal investigation.

It will thus be seen that the present presents considerable improvements upon, whilst it maintains throughout the excellent character of, the first edition. The illustrations which are particularly clear and original, have been increased from 120 to 133, and add very much to the utility of the work. The chapter and the illustrations on the subthalamie region are exceptionally good, and demonstrate the intimate knowledge which the author possesses of this most intricate part of the brain.

The English translation is most satisfactory, and we have to thank Drs. Vittum and Riggs for having made this important work of Dr. Edinger's accessible to English-speaking students of Neurology.

A. BEEVOR, M.D.

---

*Sanity and Insanity.* By CHARLES MERCIER, M.B.  
London: Walter Scott, 1890, pp. 395.

THIS work treats its subject on lines different from those usually adopted. It is not intended at all exclusively for medical men and it presumes no previous knowledge of the nervous system or of the mind. It is as far as possible free from technicalities, and is written with a fluency of style and a profusion of illustrations and analogies, very unusual in medical literature.

Its main distinction, however, from any other work on the subject, so far as we are aware lies in its way of looking at Insanity, its analysis and classification of the forms of insanity. Hitherto the classifications of insanity have been what is usually termed "artificial;" that is, they have not been based on any broad fundamental relations, but on some more or less superficial point. These classifications correspond to the botanical classification based on the number and arrangement of stamens

and pistils. Suicidal maniacs are a group of insane people, and such a grouping is for practical purposes a very useful one, but it connotes very little more than the tendency to suicide, and for purposes of study of Insanity it is almost valueless. The old botanical classification is still valuable as an index to plants, but there is no suggestiveness in it. It helps us no farther forward. Similarly the old classifications of insanity are useful still for example for asylum purposes, but they do not help us to understand Insanity. The classification attempted by Dr. Mercier is a "natural" classification. It is based on the views of Dr. Hughlings Jackson regarding Insanity or rather regarding Insanities, that they are "dissolutions" of mind, that is to say, reversals of evolution. Dr. Mercier, with a knowledge of the views

Herbert Spencer and Hughlings Jackson on the one side and a direct acquaintance with Insanity on the other side, has produced a work of real originality; a work which may not do much to help the so-called "practical man," but will do much to help those who desire some clue in the labyrinth of mental disorders, as well as some principles to guide them in dealing with those mentally disordered.

The first two chapters are devoted to an exposition of the nervous system, and the third to an exposition of mind. Medical men, as well as others, are sometimes singularly and even complacently ignorant of elementary psychology, and in consequence of this ignorance are ready to accept, and even to present mere verbal quibbles as true explanations of psychical phenomena. Careful perusal of these chapters would clear away, at least, some of the mist that surrounds the subject. The fourth chapter discusses "what Insanity is," and the last ten chapters are divided equally between the Causes and the Forms of Insanity. We have endeavoured to indicate the *differentia* of this work, and it would be useless to discuss these chapters in detail. Suffice it to say that they show breadth of grasp and knowledge of detail, not often combined; that they are suggestive more than exhaustive, and what for the medical as well as the lay reader is a matter of no small consequence, their interest never flags. Perhaps on this last point we might venture to say that if Dr. Mercier would discard an occasional flippancy from his writing, he would not weaken the vigor of his style, and would remove an element that is occasionally objectionable.

The work is, as we have said, intended for lay as well as medical readers, and we can recommend it heartily to both.

Such knowledge as it conveys regarding both Sanity and Insanity should be in the possession of every well-educated man, and of every well-educated woman also. When men and women know more than they do of both their mind and their body, the world will be the better and not the worse for it.

JAMES ANDERSON, M.D.

---

*De l'Acromégalie, Maladie de P. Marie.* Par SOUZA-LEITE, J.D. Paris: Lecrosnié et Babé.

THIS work aims at giving a full account of the present knowledge concerning the disease to which the name of "Acromégalie" was given by the distinguished clinician who first described it. Dr. Souza-Leite has brought together and analysed with the greatest care all the cases hitherto published. The chapters on the etiology, symptoms and treatment of the disease contain little more than is to be found in the paper on acromegaly published in *BRAIN* (July, 1889).

P. Marie stated then that he regretted his inability to give a full account of the anatomo-pathological characters of acromegaly, as he had performed one autopsy only. He had found in that examination that the hypertrophic process affected the spongy tissues of the bones (short bones, flat bones, epiphyses) more especially, there being at the same time hypertrophy of the vertebræ, sternum and clavicles. He stated that hypertrophy of the pituitary body, with enormous dilatation of the sella turcica, persistence of the thymus, and finally hypertrophy of the cord and ganglia of the sympathetic system, were the chief pathological appearances found at the *post-mortem* examination of acromegalic patients. Dr. Souza-Leite mentions seven *post-mortem* examinations, and, although some of these autopsies are incomplete, yet for the most part they establish on a firmer basis the accuracy of P. Marie's statements.

In the seventh chapter the author discusses the diagnosis of acromegaly from a new disease, to which P. Marie has given the name of *ostéo-arthropathie hypertrophiante pneumique*. This is by far the most important chapter in the whole book, and it will interest the readers of *BRAIN* if the author be quoted almost

verbatim. Let it be stated beforehand, however, that under the name of ostéo-arthropatie hypertrophiante pneumique is meant a peculiar deformity of the bones occurring in patients afflicted with some lung affection. According to the same author three cases have been mistaken for acromegaly (W. Erb, Ewald, O. Fraentzel, Gourand, R. Saundby), and for osteitis deformans (L. Elliott). The following lines occur in a lecture published by P. Marie in the *Bulletin Médical* (December, 1889) :—

“The hands in this disease (pneumic hypertrophie osteo-arthritis) are truly enormous. I might even say that their size is even more astounding than that of the hands of acromegalic patients, but whereas the hands of the latter patients are merely thick and stumpy, those of patients suffering from ‘pneumic hypertrophie ostéo-arthritis’ give the observer the impression of something never seen before. They are not only thick, but deformed also.

“The three segments of the hand, namely, the fingers, carpo-metacarpal region and wrist must be studied separately. Of these three segments the fingers suffer most. Sometimes they are simply a little longer, though always distinctly thicker, than normal. The circumference of the first phalanx of the radius may measure 5 cm., 7, or 10 cms. for instance. These dimensions exceed those of patients suffering from acromegaly by one centimetre. The shape of the fingers in pneumic hypertrophic ostéo-arthritis is also peculiar, in so far that the last phalanx is greatly hypertrophied, and becomes so bulbous that it is often the thickest of the three. The *fingers* therefore resemble drumsticks, the thumb being like a bell-hanger. This is never the case in acromegaly, for, in the latter disease, each of the digital segments is proportionately increased. The nails covering the extremity of the deformed finger are deformed likewise. They are considerably widened (2 cms. and more for the medius), and are longer and in-curved (Hippocratic aspect), so that the extremity of the thumb of our patient, for instance, when looked at sideways, exactly resembles the head and curved beak of a parrot. [Nothing like this is ever seen in acromegalic patients whose flattened nails appear, on the contrary, to be too small for the thick phalanges which they protect.]

“Attention must also be drawn to the very noticeable longitudinal striation of nails, to their marked tendency to split, and to the formation of longitudinal fissures. Their peripheral extremities are not infrequently of a well-marked and vivid rose colour. In three cases it was also noticed that the last phalanx could be *hyperextended* with abnormal ease.

“ So much for the fingers. The case is different with the *carpo-metacarpal part of the hand*, for its size and shape do not greatly differ from those of a normal hand, except that the heads of the metacarpal bones are slightly enlarged, so that there is a singular contrast between the size of that region and that of the fingers. . . . In cases of acromegaly, on the contrary, the metacarpophalangeal region of the hand is enormous ; I might even say that the increase in size of the metacarpophalangeal part of the hand is relatively more marked than that of the fingers ; and this deformity gives to the hand the aspect of a ‘ battoir ’ [the spade-like hand of English authors].

“ Let us now study the third segment, the *wrist*. In all cases hitherto published the widening of this region was noticed, and this condition is very noticeable on the various pictures appended to the notes of the cases. The inferior extremity of both bones of the fore-arm suddenly becomes much thicker and forms an enormous projection on the under-surface of the hand, the increase in size taking place in an antero-lateral as well as a transverse direction. The inferior part of the fore-arm thus becomes larger than the middle part, larger even than the upper part situated just above the elbow.

“ Notice also that the wrists are markedly deformed, and every unbiassed person on seeing one of these wrists for the first time, would certainly give to the disease the name of arthritis deformans. Nothing like this is ever present in acromegaly, for although the wrists of some patients are thicker than those of a normal individual, the increase is quite proportionate to the size of the upper limb, and there is at this spot no projection, no sudden widening—in one word, no deformity. This widening also is never so marked. . . .

“ If this be the aspect presented by the hands of a patient suffering from pneumatic hypertrophic arthritis, that of the feet is exactly similar. If the latter be also divided into three segments (toes, tarso-metatarsal region, malleolar region), we see that the toes, like the fingers, are not only somewhat lengthened, but markedly broadened, and that this process is more specially apparent in the last phalanx. They resemble a bell-clapper even more than the fingers do, the nails being enormous and curved in. The increase in size of the second segment (tarso-metatarsal region), like the increase in the corresponding region in the hand, is less marked than in other parts of the foot, although it is evident that its dimensions are larger than usual, and that the greatest increase takes place in the heads of the metatarsal bones.

"The size of the third segment of the foot (malleolar region), like the wrist, is enormously increased in all directions, the transverse bi-malleolar diameter is not less than 112mm., and this region, like the other segments, is evidently deformed. The lower part of the leg is therefore distinctly larger than the middle part; hence many authors have given to this deformity the name of 'elephant's foot.'

"This increase is due doubtless to the augmented volume of the bones. . . . In acromegaly, on the other hand, the soft parts themselves, like the bones, undergo hypertrophy, and in the latter disease, therefore, we no longer deal with a hypertrophy *en masse* of all the tissues of a region. The diaphyses, more especially the articular extremities of all the long bones, are notably increased in size; but this hypertrophy does not appear to be quite symmetrical in the two bones of the segment of a limb. The long bones of the distal segments of the limbs (ulna, radius, tibia, fibula) are relatively more hypertrophied than those of proximal parts of the limbs (humerus, femur). The elbows and knees are also larger than natural, the patellæ especially showing a distinct increase. It is possible also that the kyphosis before-mentioned is dependent, to a certain extent, on lesions of the intervertebral articulations.

"The patients are particularly clumsy with their hands, their elbows are in a state of more or less permanent flexion, whilst complete extension is impossible. The same remark applies to the knees also. I cannot say whether anything resembling this condition is to be noticed in the coxo-femoral articulations, but in our patient at any rate the movements of the shoulders were certainly impaired."

The last part of Dr. Souza Leite's work is a complete report of all cases of acromegaly hitherto published.

The book is profusely illustrated with 102 woodcuts, many of which are original.

M. ARMAND RUFFER.

*Small Photographic Atlas of the Nervous System: The Brain.* By Dr. J. LUYSS. Paris: F. B. Baillière, 1888.

THIS atlas is but a compendium of his large work, *Iconographie Photographique des centres nerveux*, in which the author manifested notable divergences from the generally received



opinions. Dr. Luys views with disfavour the classic description of the so-called sensory bundle, in its course through the bulb, from the posterior columns of the spinal cord to the cortex. How long, he says, can an *à priori* statement, made in the name of physiology, serve for an anatomical classification, and give a name to any nerve bundle whatever?

This criticism does not appear to us to be well founded, for anatomy cannot be considered as a science in itself, but is only one of the constituents of the whole science of the living being, which includes both morphology and the study of bodily functions. Moreover, even in descriptive anatomy we see muscles constantly receiving names derived from the part they play in the physiology of movements. Faithful to his principles, Dr. Luys gives this purely anatomical definition of the brain:—"The *ensemble* of convolutions of each lobe, connected from one side to the other (commissural fibres), and connected to the central thalamo-striated and to the sub-thalamic nuclei (white fibres: cortico-thalamic, cortico-striated, cortico-subthalamic)."

A view to which the author attaches chief importance refers to the anatomical and physiological relations between the central nuclei and the cortex. The numerous photographic illustrations which make up the second part of this Atlas (the first part of which is merely an anatomical *résumé*), and are good from the purely photographic point of view, serve the author as a basis to establish his peculiar views concerning the origin and distribution of the cerebral radiating fibres. It seems to us difficult, however, that macroscopic sections can clearly show the passage of fibres which have such varied directions, and we cannot help wondering at his reliance on this one mode of investigation. For, on the other hand, we see that Obersteiner in his classical work enumerates with details at least five methods of research, elaborated by such men as Stilling, Flechsig, Türk, Meynert, Gerlach, Gudden and Weigert.

Among the various statements of the author which appear to us difficult of admission, we find that in the brain of men of very powerful intellect Dr. Luys has noted a very large development of the convolutions in the ascending parietal region. He also reports that in cases of chronic hallucinations the para-central lobule undergoes morphological change which can be seen by the naked eye. He attributes to the region of the cuneus the property of receiving acoustic impressions. Further, the grey substance of the central regions appears to be a favourite subject of the author's who has discovered a nucleus, an accessory branch of



the red nucleus of Stilling, called by Forel *Corpus Luysii*. In his opinion the nucleus of Stilling is a region which is an essentially motor and excitable region; and the optic thalamus contains nuclei to which he attributes special physiological properties—olfactory, visual, &c. But one of the reasons given by the author in favour of his point of view appears rather contradictory, for he says that the white fibres of the corona radiata, which he assumes to be connected with his visual nucleus, are distributed to the antero-lateral convolutions. Dr. Luys thereby implicates that optical impressions are produced in these portions of the cortex; whereas the cortical centre for vision is usually localised in totally different parts of the brain surface, notably in the occipital lobes. The functions of these latter Dr. Luys makes out to be auditory, on the ground that he thinks he has traced into them radiating fibres from the acoustic nucleus he believes to exist in the thalamus. Finally, he ascribes trophic functions to the central thalamic region, which he describes as connected with olfactive and optic fibres.

The optic thalamus would thus be a receptive and elaborative centre for sensory impulses from the periphery on their way to the cortex. He also attributes a peculiar structure to the corpus striatum, which he describes as containing, besides its numerous polygonal or oval-shaped cells, small yellowish corpuscles infinitely ramified which appear to proceed from cerebellar elements. The yellow nucleus which he afterwards describes, the innermost portion of the lenticular nucleus, would thus bear a trophic relationship to the lobes of the cerebellum.

Again, according to Dr. Luys, the so-called converging fibres, which are in connection with the central nuclei, begin in the middle of the intercellular network of the grey cortex, and consequently are not in direct communication with the nerve cells; and the bulk of the fibres composing the internal capsule does not unite directly the cortex with the medulla, but ends in the optic thalamus. The corona radiata would thus be composed of fibres arising from the central nuclei, the corpus striatum being motor, the optic thalamus sensitive and sensorial. These, in brief, are the chief peculiarities of Dr. Luys' teachings. We see that if the opinions put forth in this work deserve to be characterised as original, they are far from being in consonance with the statements of recent authors, such as Obersteiner, Edinger, and other recognized authorities in nerve anatomy.

P. CHASLIN, M.D. (Paris).

## Abstracts of British and Foreign Journals.

**Current Nerve Anatomy and Physiology.**—Titles and Indication of Contents of recent Papers. By ALEX HILL, M.D., Master of Downing College, Cambridge.<sup>1</sup>

### ANATOMY TEXT-BOOKS.

1. Manuale di anatomia degli organi nervosi centrali dell'uomo. MINGAZZINI (*Rome*, 1889).
2. The Anatomy of the Central Nervous Organs in Health and in Disease. OBERSTEINER AND HILL (*London*, *Griffin & Co.*, 1890, pp. 432, figs. 198).
3. Twelve Lectures on the Structure of the Central Nervous System. EDINGER (*translated from the second edition by VITTMUM AND RIGGS.*) *Davis, Philadelphia and London*, 1890, pp. 230, figs. 133.
4. Report upon the Progress of the Anatomy of the Central Nervous System during the year 1889. EDINGER (*Schmidt's Jahrbücher*, cccxxviii., p. 73, 1890.)

### ANATOMY.

5. Two Chinese Brains. DERKUM (*J. of Nervous and Mental Disease*, xiv., p. 421, 1889).

### TOPOGRAPHY.

6. The Topographical Relations of the Brain to the Skull-case. MÜLLER (*Bern*, 1889, pp. 60, plates ii., figs. 8).

### COMPARATIVE ANATOMY.

7. The Central Nervous System of the Cetacea. KÜCKENTHAL AND ZIEHEN (*Denksch. der Med.-Naturw. Gesellschaft zu Jena*, iii., pp. 80-200, plates iv.-xii., with tracings in outline of the life-size drawings).

<sup>1</sup> To my great regret I have been unable to find time to make these abstracts complete to the end of the year 1890. I shall hope to fill up the gaps in the next number of BRAIN.—A. H.

## MORPHOLOGY.

8. The Origin of Vertebrates from Arachnids. PATTEN (*Q. J. Micros. Anat.*, Aug., 1890, pp. 317-379, plates xxiii. and xxiv.)

This paper contains an elaborate description of the nervous system of the scorpion. The author contends that its cephalothoracic neuromeres, nerves, sense organs and mesoblastic somites, present, in a general way, not only the same specialisation and the same numerical arrangement in groups, but also the same difference as a whole from the body-segments as do the corresponding parts in the vertebrate head.

9. On the Origin of Vertebrates from a Crustacean-like Ancestor. GASKELL (*Q. J. Micros. Science*, cxxiii., August, 1890, pp. 379-445, pl. xxv.-xxviii.)

Several morphologists have been led to the conclusion, from a study of the vertebrate brain, that the anterior end of the alimentary canal must, in vertebrate ancestors, have pierced the floor of the third ventricle. Gaskell goes further, and says that the cavity of the central nervous system is the primitive gut, the ventricles of the brain the primitive stomach. Hence he traces the vertebrate limb of the animal tree back to a meeting point with invertebrate limbs in a proto-arthropod, not, as most morphologists suppose, in a proto-annulose animal. (*For an account of his theory see BRAIN, July, 1889, p. 1.*) The present paper sets forth the evidence obtained from a study of the central nervous system of young lampreys (the ammocoetes of *Petromyzon Planeri*). In these animals the ventricular cavity is large; its roof formed by a much folded membrane. If the membrane were unfolded a large cavity similar to a crustacean stomach would be produced. The resemblance of the infundibulum to the œsophagus of the crustacean, compressed from above downwards, is more marked than in the sheep (see previous paper). Traces are also visible of another diverticulum from the back of the third ventricle, compressed from side to side, the *conus post-commissuralis* (? homologous with the *ganglion interpedunculare*); this Gaskell regards as the altered duct of the crustacean liver. It ends in a cellular mass which invests the greater part of the brain, and consists of cells, like liver-cells, separated from one another by pigment. The pigment, Gaskell thinks, indicates the former presence of abundant inter-cellular blood-vessels. The ganglia *habenulæ* are large, the right being the larger. They are connected respectively with the large dorsal and small ventral pineal eyes,

and a large right and small left Meynert's bundle, and represent the optic ganglia of the pair of median eyes of the crustacean-like ancestor. The pineal eye is of the invertebrate type. The portion of the brain-case which covers it corresponds to the cuticular lens of invertebrates.

The brain is made up of a lining epithelium of fattily-degenerated columnar cells, in places ciliated; substantia gelatinosa centralis containing the tail-like processes of columnar cells, and nerve matter. The raphe is formed by the fusion of apposed layers of degenerated columnar cells. The prechordal and epichordal portions of the central nervous system (parts in front of and behind the infundibulum) agree, in minute structure, with the supra- and infra-oesophageal ganglia of the Crustacea, with which they respectively correspond. The chief difference between them depends upon the much greater number of small nerve cells, grouped in masses, in the prechordal (supra-oesophageal) than in the epichordal (infra-oesophageal) portion.

10. Address on the Origin of the Vertebrate Nervous System.

GASKELL (*Brit. Med. J.*, No. 1563, pp. 1341-1345, figs. 1-10, Dec. 13, 1890).

#### EMBRYOLOGY.

11. Development of the Human fore-brain from the end of

the first to the beginning of the third month. HIS (*Abhandl. der math.-phys. Classe der K. Sächsischen Gesellsch. der Wissensch.*, vol. xv., No. viii., pp. 675-736, figs. and plate, Nov., 1889, *Leipsic, Hirzel*).

12. The Development of the Ganglionic System in the Fowl.

GOLOWINE (*Anat. Anz.* V., No. 4).

13. A Stage in the Growth of the Primate Brain. CUNNINGHAM

(*Brit. Med. J.*, Sept. 20, 1890, p. 683).

The transitory infoldings of the thin cerebral wall which appear towards the end of the second month explained as due to a want of harmony in growth between the brain case and the brain, and especially to the tendency on the part of the brain to grow backwards into an occipital lobe.

14. Address on Cerebral Anatomy. CUNNINGHAM (*Brit. Med.*

*J.*, Aug. 2, 1890, p. 277).

The morphological value of the fissures and convolutions. Difficulty in harmonizing ontogeny and phylogeny. Convolutions, homoplastic, if not homogenetic. The transitory fissures of the

third month bear no relation to the fissures of the adult brain, and are probably peculiar to the primate brain. The early appearance of annectant gyri a characteristic human feature. Hence in Man some fissures, the Rolandic for example, begin their existence as separate pieces, which extend towards one another until they meet over the deep annectant gyri, or fail to meet, as the case may be. Critical account of the different mechanical explanations of the formation of convolutions, and conclusion that they cannot be attributed to the influence of external forces, but are due to such internal tendencies to growth as determine the form of other physiologically distinct organs.

### PHYSIOLOGY.

15. The Function of the Central Nervous System in Invertebrate Animals. STEINER (*Sitz. d. K. P. Akad zu Berlin*, 1890, ii., p. 39).

A long series of observations on the functions of the several ganglia in various classes of invertebrates.

16. Voluntary and Reflex Muscular Contraction. HAYCRAFT (*J. of Physiology*, xi., 4 and 5, pp. 352-369, pl. xii., July, 1890).

### PHYSIOLOGY: COMPARATIVE.

17. The Comparative Physiology of the Great Brain. SCHRADER (*Deutsche Med. Wochenschrift*, No. 15, 1890).

Investigation of the function of the bird's brain, using for experiments the owl and hawk as presenting a higher type of brain, than the dove and hen which have hitherto been the favourite subjects. The right hemisphere was removed in the owl, which became lame with its left foot and blind with the left eye, although flight was not affected. In the hawk both frontal lobes ("frontalhirne") were removed, and the operation resulted in complete spastic paralysis of the legs, while wings and tail acted normally. The paralysis subsequently disappeared completely. It appears from these experiments that whereas the limb muscles are not represented in the cerebral hemispheres of the dove or hen, birds of prey approach mammals so far as the cerebral representation of the hinder extremities is concerned.

## PHYSIOGENY.

18. The Effects which follow Destruction of different Parts of the Brain of New-born Animals, and the Development of the Functions of the same. BECHTEREW (*Medizinskoje Obosrenje*, No. 4, 1890).

The effects of destruction of a portion of the brain of a new-born animal are similar to those which follow the same operation in the adult, provided the fibres of this part are already medullated. No result is obtained if the fibres are still without medullary sheaths. Destruction of the motor area or section of the crus cerebri in a blind puppy are without effect, whereas section of the already-medullated restiform body produces rolling movements. The guinea pig is more highly-developed at birth, and injury to its brain at this time produces the same results as in the adult animal. Bechterew investigates the successional development of sensory and motor functions in the cortex.

## PSYCHOLOGY.

19. The Duration of Memory-pictures. PANETH AND EXNER (*Centralbl. f. Physiol.*, iv., 3, pp. 81-83, 1890).

Sensory impressions give rise to "primary memory-pictures." When attention is directed to these pictures projected upon our consciousness they are fixed as secondary memories. If, owing to pre-occupation, attention is not directed to the primary memory, it rapidly fades. The paper considers the duration of this primary memory.

20. Psycho-Physiological studies in Protista. VERWORN (*Jena, Gustav Fischer*, 1889. *Physiol. Centralbl.*, iv., 4, 1890, pp. 103-108).

## CEREBRUM: CORTEX.

21. The Origin of the Convolutions of the Great Brain. SCHNOPFHAGEN (*Leipsic, Deuticke*, 1891, pp. 122, figs. 18).

Description of the white matter of the cerebrum, as investigated by de-fiberizing. Conclusions from the disposition of the fibres, as revealed by this method, as to the causation of convolution.

22. Intraparietal Sulcus. CUNNINGHAM (*J. Anat. and Phys.*, xxiv., pp. 135-155, 1890).

Of sixty-two hemispheres examined only four exhibited an intraparietal sulcus divided into three separate parts. The disposition usually considered as normal (ramus verticalis superior distinct from the confluent rami horizontalis et verticalis inferior) only occurred in ten. The united vertical and the horizontal rami were distinct in ten. The three parts of the sulcus were confluent in thirty-five. The lower vertical limb was confluent with the horizontal limb, the upper vertical being independent, in two. The ramus occipitalis was quite distinct from the ramus horizontalis in thirty-six, and in ten others they were partially separated by a bridging convolution. The sulcus occipitalis transversus of Ecker is regarded as belonging to the intra-parietal system, and not, therefore, homologous with the external parieto-occipital fissure of apes.

#### CORTEX : PHYSIOLOGY.

23. Changes in Excitability in the Psychomotor Cortex.  
LANDOIS (*Verhandl. der Medic. Vereins zu Greifswald*, 1888-1889).

24. Partial Cerebral Atrophy of Peripheral Origin. SIBUT  
(*Paris, Baillièrre et Fils*, 1890, pp. 84).

A collection of recorded cases in which the cortex was found to be atrophied as the result of infantile paralysis or amputation of a limb. Conclusion: the atrophy is the more marked the younger the subject at the time of the peripheral lesion, and the longer the period which has elapsed since the lesion; it is due to functional atrophy, not degeneration; it affects the motor centre of the limb affected; the centre for the upper limb lies in the marginal (transverse) convolutions at the junction of their upper and middle thirds, and by preference in the ascending parietal convolution; the centre for the lower extremity is situate in the upper part of these same marginal convolutions and in the paracentral lobule; it usually occupies the ascending frontal. Histologically the atrophy consists in a diminution of pyramidal cells.

25. Descending Degenerations which follow lesions of the Gyrus Marginalis and Gyrus Fornicatus in Monkeys, with appendix on Degenerations which follow removal of the External Motor Cortex. FRANCE (*Phil. Trans. Roy. Soc.*, 180, B, pp. 331-354, plates 27-29, Jan. 15, 1890).

Horsley and Schäfer had found that stimulation of the gyrus



fornicatus gives rise to no muscular contractions; its removal is not followed by paralysis, but does cause deficiency in general and tactile sensibility of the opposite side of the body. France finds that removal of either the marginal or the fornicate gyrus is followed by descending degeneration of the opposite lateral pyramidal tract, traceable to the lower lumbar region. When the marginal gyrus has been removed the degeneration in the cord is confined to the portion of the tract which borders on the direct cerebellar tract. After removal of the fornicate gyrus it occupies the whole sectional area of the lateral pyramidal tract.

26. The Visual Area on the Surface of the Hemisphere.

BECHTEREW (*Archiv psichiatrii, neurologii, &c.*, No. 1, 1890, in *Russian*. *Neur. Centralbl.*, 1890, p. 237, *abstract*).

Digest of results of various observers criticised in the light of Bechterew's own observations. He finds the area widely extended, including the whole occipital and part of parietal lobes and reaching in a coronal direction from the temporal lobe over on to the median surface. It is divisible into two great regions, of which one presides over the retina of the same side, and the other over the retina of the opposite side.

27. The Physiology of the Movements and Convulsions liberated by the Cerebral Cortex. KORANYI AND TAUSZK (*Internat., Klin. Rundschau*, No. 14, iv., 1890).

Having exposed a cerebral hemisphere in the rabbit, they painted the surface with Liebig's extractum carnis, by which means clonic contractions of the face muscles are provoked, but not general convulsions. During this state of excited activity, however, stimulation of the skin of the face provokes contraction of the right fore limb, and when stronger of the hind limb, and then of the opposite side of the body. The face is, in fact, an epileptogenous zone. To determine whether the cortex is merely a node in the reflex arc, the authors removed portions of its substance and eventually the whole hemisphere, but found that while at first the reflex convulsions were in this way inhibited, they could after a time be obtained in full force. They conclude that the convulsions are obtained through a reflex mechanism in the mid-brain.

28. The Croonian Lectures on Cerebral Localization.

FERRIER (*British Medical Journal*, Nos. 1536-1541, June 7 *et seq.*, 1890, with illustrations).



29. Sight Area and Eye Movements. MUNK (*Sitz. d. K. P. Akad., Berlin*, Jan. 16, 1890). See BRAIN, part 49, 1890.

30. The first Temporal Convolutions, right and left, in a Man Deaf with the Left Ear. MANOUVRIER (*Bull. de la Soc. de Psychologie Physiologique*, 1889, p. 24).

Bertillon, the statistician, was deaf with his left ear (from ten years old) and left-handed. The right upper temporal convolution was notably smaller than the left. As is to be expected in left-handed people, Broca's convolution was better developed on the right than on the left side. A noticeable difficulty in speech which he exhibited may have been due, therefore, to his speaking with the deaf hemisphere. The optic region on the right side was considered as showing a compensatory development.

#### CORTEX : CONDUCTING PATHS.

31. The Central Convolutions a Central Organ for the Posterior Columns. FLECHSIG AND HÖSEL (*Neur. Centralbl.*, July 15, 1890, pp. 417-419).

Flechsig has already taught on developmental grounds that tracts of fibres connect the posterior columns with the cortex of the posterior central convolution *via* the " tegmental radiations " and fillet. Authors now describe a case in which paralysis of fifty-two years' standing (commencing in the second year) is associated with porencephalia in the region of the central convolutions. Atrophy of the left posterior central convolution, part of lobulus paracentralis, medullary substance under the upper third of the anterior central and front of upper parietal convolution is accompanied by atrophy of the left pyramidal tract and also of the fillet-layer (of the pons especially), left interolivary layer, fibræ arcuatæ internæ, and the nuclei funiculi cuneati, and to a less extent funiculi gracilis. The left red nucleus and right brachium conjunctivum cerebelli and nucleus dentatus are also small.

#### CORTEX : HISTOLOGY.

32. The Brain-Cortex and the Central Origin of the Nerves. MARTINOTTI (*Intern. Monatschr. f. Anat. und Physiol.*, vii., 2, p. 69).

The writer is of opinion that the course of nerve-fibres can be rendered evident by the use of Golgi's method, and that protoplasmic and nervous processes can be distinguished. Protoplasmic

processes are never in connection with the nerve network, but join neuroglia cells. Nerve cells can be distinguished (functionally) according as they are directly or indirectly connected with nerve-fibres. The former belong to motor, the latter to sensory, regions.

#### CORPUS CALLOSUM.

33. The Effects of Section of the Corpus Callosum. KORANYI (*Pflüger's Archiv.*, xlvii., pts. 2 and 3, 1890).

The corpus callosum was cut through in dogs. When successfully accomplished the operation gave rise to no obvious disturbance of movement. If the hemisphere was injured temporary disturbances of vision, common sensation and movement resulted; as they did also if the white matter was injured even at some distance from those portions of the cortex in which these functions are said to be localised. The results of these accidental injuries are detailed. Section of the corpus callosum may give rise to general convulsions.

34. The Brain without Corpus Callosum. JELGERSMA (*Neurol. Centralbl.*, No. 6, pp. 162-167, March 15, 1890).

Pathological evidence in favour of Jelgersma's theory as to the cause of convulsion in the cortex of the cerebrum and cerebellum (*cf. these abstracts*, BRAIN, 1889, p. 410).

#### STEM-GANGLIA.

35. The Physiology of the Infracortical Ganglia and their connection with Epilepsy. ZIEHEN (*Arch. f. Psychiatric*, xxi., 1890).

Pricking, weak faradization and section used as stimuli to the ganglia in rabbits exposed by removal of the hemispheres and fornix. Stimulation of the nucleus caudatus gave rise to such contractions as result from stimulation of the anterior part of the motor region of the cortex. No nodulus cursorius in Nothnagel's sense was found. From the nucleus lenticularis the same effects were obtained. Stimulation of the optic thalamus produced contractions on both sides of the body. Section at back of thalamus (especially across the tegment through the internal geniculate body) gave rise to running movements and cries. Movements also resulted from stimulation of the corpora quadrigemina. The author looks upon these movements as reflex; the sensory tract including the optic tract, auditory nerve, &c. The pyramidal tract was not

the motor tract for these movements. Tetanic contractions are characteristic of the infra-cortical ganglia as contrasted with the clonic spasms of cortical origin. Hence Ziehen concludes that the tonic components of the epileptic fit are liberated not from the cortex but from the stem-ganglia.

36. Effects upon the Bodily Temperature of Lesions of the Corpus Striatum and Optic Thalamus. WHITE (*J. of Physiology*, x., 1 and 2, pp. 1-25, Jan., 1890).

A variety of control-experiments performed to make sure that no cause, except the injury to the grey matter, causes rise in temperature. All precautions being taken, it was found that in rabbits the body temperature is raised on both sides of the body after unilateral injury to the corpus striatum or optic thalamus. Injury to the white matter in the neighbourhood of these grey masses produced no result.

#### PES PEDUNCULI.

37. The Fibre System of the Pes Pedunculi and Cortical Relations of the Corpus Geniculatum Internum. ZACHER (*Neur. Centralbl.*, July 15, 1890, p. 440).

#### MID-BRAIN.

38. The Origin of the deep Medullary Substance of the Region of the Corpora Quadrigemina. HELD (*Neur. Centralbl.*, Aug. 15, 1890, No. 16, pp. 481-483).

Developmental study in kittens and young rats after Flechsig's method.

#### MID-BRAIN: FILLET.

39. The Physiology of the Fillet (a case of Glioma of the Posterior Horn). ROSSOLYMO (*Arch. f. Psych. u. Nervenkrankh.*, xxi., 1890).

Great disturbance of sensibility; degeneration of the posterior roots, and diminution in the number of fibres in the right inter-olivary layer, pyramid, fillet, pons and crus cerebri. Goll's column unaffected. Conclusion: that the last-mentioned has no immediate connection with the posterior roots; that the fillet is made up chiefly, if not entirely, of fibres which convey skin-sensibility from the opposite side of the body.

## MID-BRAIN: CORPORA QUADRIGEMINA.

40. The Connections between the Posterior Tubercles and the Auditory Nerve. FLECHSIG (*Neurol. Centralbl.*, 4, Feb. 15, 1890, pp. 98-100).

Fresh observations, in conjunction with Held, on the brains of kittens, which (*contra* v. Monakow) confirm the author in his views as to the connection of the ramus cochlearis with the posterior tubercles *via* the anterior auditory nucleus and corpus trapezoideum. Flechsig's former observations were made on human brains. In the cat at birth the ramus vestibularis is not yet myelinated. Its development shows that the primary cochlear centre gives rise to four systems of fibres at least. Three of these take part in the formation of the corpus trapezoides, while the fourth crosses partially behind the latter in the raphe and joins it in the neighbourhood of the upper olive. The lateral fillet is connected with the ramus cochlearis by constituents of the corpus trapezoides, and also by these fibres which cross behind it. The latter come chiefly from the tuberculum acusticum, the former from the anterior nucleus.

In Man the central auditory apparatus is less developed than it is in the lower animals. Flechsig thinks it not impossible that the striæ acusticæ (which are first myelinated some time after birth) are connected with the posterior tubercles.

## MID-BRAIN: OPTIC LOBES.

41. Time Relations of Stimulation of the Optic Lobes of the Frog. WILSON (*J. of Physiology*, xi., 6, pp. 504-508, Nov. 1890).

Delay of stimulus in optic lobes determined at '02," being exactly the same as in the case of the cerebral hemisphere. Curve of movement resembled the characteristic cerebral cortical curve. Conclusion: that the cells in the optic lobes of the frog have motor connections similar to those of the cerebral cells in the mammal.

## CEREBELLUM: FUNCTION.

42. The Function of the Cerebellum. GOWERS (*Neur. Centralbl.*, No. 7, 1890, pp. 194-5, April 1).

A condensed account of a theory of the function of the cerebellum which can hardly be still further abstracted. In outline

it is as follows :—A distinction has to be made between the central lobe of the cerebellum and its hemispheres. The former presides over co-ordination of movement, perhaps acting through the cortex of the hemispheres. The two conducting paths connected with the cerebellum (lateral cerebellar tract and posterior column) conduct upwards. These tracts are apparently ascending continuations of muscular nerves. The impulses which ascend these tracts are not, as a rule, sensible, although they govern the position of the body. Impulses also ascend from the acoustic nerve, and similarly the organ receives information as to the eye-ball movements. We do not know that a single fibre descends from the cerebellum to the spinal cord. Routes are known, on the other hand, through which an influence may be exerted directly or indirectly upon the grey matter of the optic thalamus or corpus striatum.

From these anatomical data Gowers concludes: that the middle lobe of the cerebellum is a regulating centre for such centripetal impulses as have a special relation to movement; that the cerebellum exercises a controlling influence upon the cells of the cortex cerebri, just as the motor cortex controls the centre for muscle reflex upon which the so-called tendon-phenomenon depends.

This explanation of the function of the organ serves to explain the loss of patellar reflex in tumour of the middle lobe of the cerebellum; the diminution of patellar reflex in such diseases of the cerebellar hemispheres as interrupt the route from the cerebellum: many of the phenomena of athetosis and cramps in cases of disease of the cerebellum, and the peculiar relation between the symptoms of disease of the middle lobe and those of tabes—ataxy in each case being due to disease of ascending muscle-nerves.

43. Functions of the Cerebellum. BECHTEREW (*Medizinskoje Obosrenje*, 13-14, 1890).

44. The Question of the Function of the Cerebellum. BECHTEREW (*Neurol. Centralbl.*, June 15, 1890, No. 12.)

A criticism of the theory as to the function of the cerebellum developed by Gowers in the same journal, No. 7. (See *supra*.) Bechterew particularly combats Gowers' statements as to the limited connections of the cerebellum, and the absence of descending spinal tracts.

## CEREBELLUM: HISTOLOGY.

45. The Nervous Fibres of the Granular Layer of the Cerebellum and the Evolution of the Cerebellar Elements. RAMON Y CAJAL (*Intern. Monatschr. f. Anat und Physiol.*, vii., part 1).

## NERVES: CRANIAL: II.

46. The Crossing of Nerve Fibres in the Chiasma Nervorum Opticorum. DELBRUCK (*Arch. f. Psychiatrie*, xxi., 1890).

The right uncrossed bundle alone was normal, and hence the subject presented a good opportunity of studying the disposition of this tract. In the nerves its fibres are collected into thick bundles, and lie on the lateral side. In the chiasm and tract they are mixed up with the crossed fibres. They are not, however, scattered uniformly over the tract, but, at first, leave the inner and under quadrant free, and farther back a continually contracting border-zone. Retrospect of the literature.

## OPTIC CONNECTIONS.

47. Basal Hemianopsia. REMAK (*Neurol. Centralbl.*, 5, Mar. 1, 1890, pp. 130-136).

## NERVES: CRANIAL: III.

48. The Anatomy of the Oculomotor Centres in Man. PERLIA (*v. Graefe's Archiv. fur Ophthal.*, xxxv., 4, p. 287).

Perlia divides the groups of cells from which the nerve originates into two large groups, of which the hinder is the larger. Each of these is again made up of small divisions (A), the hinder or chief group comprising: (1) hinder ventral, (2) hinder dorsal, (3) anterior ventral, (4) anterior dorsal, (5) central, (6) mediolateral (Edinger-Westphal small celled group); the anterior (B) comprises: (1) the lateral anterior nucleus, (2) the median anterior double nucleus.

Fibres from the oculomotor nuclei run into the posterior longitudinal bundle, which also contains fibres from the ansa lenticularis. A network of fine fibres in the central grey matter is also in connection with this bundle.

49. The Number and Calibre of the Nerve Fibres of the common Oculomotor Nerve in the New-born Kitten and adult Cat. SCHILLER (*Comptes rendus*, cix., 14).

Number of fibres about 3,000, and so nearly the same in the kitten and cat that Schiller concludes that there is little or no accession in extra-uterine life. The finer fibres in the new-born animal are easily overlooked.

Size: In new-born kitten,  $1.5-2\ \mu$ ; at one month old,  $4-6\ \mu$ ; at one year,  $6-12\ \mu$ ; at eighteen months,  $6-12\ \mu$ .

#### NERVES: CRANIAL. VIII.

50. The Origin and Central Course of the Auditory Nerve in the Rabbit and Cat. BAGINSKY (*Virchow's Archiv*, ix., 1, p. 81).

The cochlea was destroyed in a kitten at birth, and brain examined after the animal had lived eight weeks. The posterior root, with its intercalated nerve cells, second and third layers of the tuberculum laterale, anterior auditory nucleus, corpus trapezoides, striæ medullares and superior olive were atrophied. After the crossing in the corpus trapezoides the atrophy was continued through the inferior fillet into the posterior brachium, and the ventral medullary layer of the posterior corpus quadrigeminum. In the cat the larger mesial part of the posterior auditory root goes into the anterior nucleus, the smaller lateral part into the tuberculum laterale. The outer and inner nuclei were intact. So, too, was the mesial geniculate body. (*See also these abstracts*, BRAIN, 1889, p. 421).

#### NERVES: CRANIAL: V.

51. Hemiatrophia Facialis and the Origin of the Trigeminal Nerve. HOMÉN (*Neur. Centralbl.*, July 1 and 15, 1890, pp. 385-388 and 430-438).

A carcinoma of the dura mater pressed upon the Gasserian ganglion, the trigeminal nerve and its branches. The resulting atrophy of the roots of the nerve was observed microscopically. The degeneration backwards affected, chiefly, at least, the portio major in the pons, and of the several internal roots of the nerve the "ascending" was atrophied; so too was the cerebellar root. The "descending" root was less affected. No alteration was visible in the root from the substantia ferruginea. The cells were diminished in the sensory nucleus.

Certain degenerated fibres were found in the facial nerve, due apparently to atrophy of the great superficial petrosal nerve. The cornea was cloudy and ulcerated.

## SENSES : SEMI-CIRCULAR CANALS.

52. Motor Disturbances after removal of the Semi-circular Canals. EWALD (*Centralbl. f. d. Med. Wissench.*, 1890, p. 114.)

After removal of the utricle, ampullæ and canals in the dove, the animal suffered from diminution in muscular power on the same side, particularly shown in the weaker movements of the wing when it attempted to escape from being held. This weakness the author attributes to the abolition of sensory impulses normally originating in the labyrinth.

## MEDULLA : ARCIFORM FIBRES.

53. The Course of the Fibræ Arcuatæ Externæ Anteriores. PAL (*Stricker's Arbeiten*, Vienna, 1890).

These fibres come out from the raphe, bend round the pyramid, and enter the corpus restiforme. At the level of the upper third of the olive, the bundle coming out of the raphe gives fibres to the pyramid, only a portion of its fibres going on to invest the olive. Some of these fibres are nevertheless continued through the pyramid to the olive by a roundabout course.

## SPINAL CORD : WHITE COLUMNS.

54. Number and Distribution of the Medullated Fibres in the Frog's Spinal Cord. GAULE (*Abh. der Math.-Phys. Classe d. K. sächs. Gesellsch. d. Wissensch.*, xv., 9, pp. 737-780, 10 plates, 1889).

The number of fibres in the white matter of the cord was counted at five different levels, viz.: at the junction with the medulla; in the middle of the cervical swelling; in the middle of the dorsal cord; at the beginning of the lumbar swelling, and beneath the ninth nerve. An attempt is then made to allot the fibres to peripheral nerves, using the tables drawn up by Birge for the fibres in the anterior and posterior roots. Gaule's figures tally with Birge's on the supposition that each root-fibre is connected with eight spinal fibres. He considers the bearing of this determination, that each root-fibre is in relation with a fixed number of cord-fibres, upon his view as to the position of the cell in the animal economy.

## SPINAL CORD : STIMULATION.

55. Muscle-contraction following Stimulation of the Spinal Cord and the Brain with interrupted Electrical Stimuli.



HAYCRAFT (*Centralblatt f. Physiol.*, iv., 6, 1890, pp. 153-4; see also *Journal of Physiology*, vol. xi., p. 352).

#### SPINAL CORD : NUCLEI.

56. The Nucleus of Origin of the Nerve which innervates the M. quadriceps cruris. LEHMANN (*Dissertation, Würtzburg*, 1890).

Extirpation of the muscle in the new-born animal resulted in atrophy of the grey matter of the cord between the fifth and seventh lumbar nerves (corresponding to 2-4 lumbar in Man). The attempt to use the results for the purpose of deciding as to the true reflex nature of the "patellar reflex" was unsuccessful.

#### SPINAL CORD : POSTERIOR COLUMNS.

57. The effects which follow Section of the Posterior Columns of the Spinal Cord and the relation of these Columns to Equilibration. BECHTEREW (*Archiv. f. Anat. und Phys. Phys. Abth.*, v. and vi., pp. 489-505, 1890).

Author is convinced that the fibres of Goll's column originate in the cells of the posterior horn, and partly also of Clarke's column, while the shorter (segmental) fibres of Burdach's column originate almost exclusively in the cells of Clarke's column. In the experiments here recorded he cut the posterior columns in the upper cervical region in doves, rabbits and dogs. The disturbance of equilibrium was never so pronounced as is the case when the inferior cerebellar peduncles are divided. From this the author concludes that centripetal impulses which determine the action of the cerebellum as an organ of equilibration, come from other parts of the cord beside the nuclei of the posterior columns, presumably the lateral cerebellar tract.

#### SPINAL CORD : CONUS MEDULLARIS.

58. Disease of the Conus Medullaris and Cauda Equina of the Spinal Cord. BECHTEREW (*Wratsch*, No. 39. Sep. 27, 1890).

#### PYRAMIDAL TRACTS.

59. Relative Development and various Dispositions of the Pyramidal Tracts in Man and Animals, and on the

Nutrition of these Tracts. BECHTEREW (*Medizinskoje Obosrenje*, Nos. 13-14, 1890).

#### ANATOMY: CONDUCTING PATHS.

60. Doubled Crossing of Cerebro-spinal Conducting Paths.  
UNVERRICHT (*Neur. Centralbl.*, No. 16, pp. 483-487,  
August 15, 1890).

Stimulation of the brain of a dog between the centres for the extremities caused flexure of the spinal column to the same side. The author set himself to decide whether this is a case of homo-lateral innervation or of recrossing. The spinal cord was cut half through, either just below the skull or in the upper dorsal region. In either case the curvature to the same side as the stimulation resulted, although the spinal cord on this side of the body was cut across.

In a number of dogs the spinal cord was divided on one side and the animal observed as long, subsequently, as possible. With the paralysis of the hind foot on the same side was associated a condition of the trunk which could only be explained by supposing that the contra-lateral musculature was paralysed. If the cord had been divided on the left side the animal maintained its trunk in a curve concave towards the left.

#### SPINAL CORD: HISTOLOGY.

61. Outlying Nerve Cells in the Mammalian Spinal Cord.  
SHERRINGTON (*Proceedings of the Roy. Soc.*, Jan. 30, 1890)

A study of the nerve cells which are scattered throughout the white columns. In the anterior columns they are multipolar, and lie for the most part amongst the fibres which join the anterior commissure. In the lateral columns spindle-shaped cells are scattered about in the neighbourhood of the intermedio-lateral tract, near the processus reticulares and (especially in the lumbar cord) near the substantia Rolandi; the cells last-named appear to be connected with the fibres of the posterior roots. Cells of Clarke's column,  $70\mu$  in diameter and of broadly oval form, are found in the posterior columns, especially in Man. They are placed with their long axes in the direction of the fibres which enter Clarke's column from Burdach's column.

62. Out-lying Nerve-cells in the Mammalian Spinal Cord.  
SHERRINGTON (*Phil. Trans. Royal Soc.*, 181, 1890,  
B. pp. 33-48, pl. 3, 4).

Description of cells which are scattered in the anterior, lateral and posterior white columns. No group of cells in the lateral column which can be regarded as the homologue of the group found at the periphery of the cord in the lateral column of the alligator, &c., but a sub-pial thickening or septum which separates the lateral cerebellar from the antero-lateral ascending columns seems to indicate its situation. Cells in the radicular zone of postero-external column looked upon as belonging to Clarke's column, and suggestion that the solitary cells in the median limb of the substantia gelatinosa are also equivalent to cells of Clarke's column. Similar outlying cells on median side of cuneate nucleus. These and cuneate nucleus (*not* vagus nucleus) regarded as belonging to Clarke's column.

#### REFLEX ACTION.

63. The Knee-jerk after Section of the Spinal Cord.  
REICHERT (*Journal of Nerv. and Mental Disease*, xv.,  
p. 71, 1890).

In twelve dogs in which the spinal cord was cut across in the cervical or upper dorsal region it was found that the most varied peripheral stimuli had no appreciable effect upon the strength of the tendon reflex. From this it is concluded that the fluctuations in extent of movement due to stimulation of other sensory nerves depends upon cerebral action.

64. Knee-jerk and its Physiological Modifications. BOWDITCH and WARREN (*J. of Physiology*, xi., 1 and 2,  
pp. 25-65, Jan., 1890).

#### REFLEX: CRIES.

65. Reflex Cries in Animals. LABORDE (*C. R. Soc. de Biologie*, 1890, pp. 83-84. Feb. 15.)

After rapid removal of the brain as far down as the pons (in guinea-pigs) reflex cries follow with perfect regularity whenever the foot is pinched.

#### NERVES: PERIPHERAL: SYMPATHETIC.

66. The Innervation of the Stomach. BECHTEREW and MISLAWSKI (*Neurol. Centralbl.*, 7, April 1, 1890,  
pp. 195-199).

67. Central and Peripheral Innervation of the Intestine.  
BECHTEREW and MISLAWSKI (*Arch. f. Anat. u. Phys. Phys. Abth., Supplement*, 1889).

Contractions of the gut registered by a manometer connected with a gutta-percha ball, included in the intestines of the dog. Conclusion that the normal tone, the rhythmic contractions and the slower peristaltic movements depend upon a local nerve-apparatus. The vagi chiefly innervate the small intestine and the upper segment of the large intestine. They contain more motor than inhibitory fibres. The splanchnics are chiefly, but not exclusively, inhibitory. Stimulation of the sigmoid gyrus or of the optic thalamus sometimes induced contraction, sometimes quiescence.

68. Development of the Sympathetic Nervous System in Mammals. PATERSON (*Phil. Trans. Royal Society*, vol. 181 (1890), pp. 159-186, pl. 22-30).

This monograph commences with some account of the views held as to the physiology, morphology and embryology of the sympathetic system; the two views with regard to its development (the subject upon which the author's investigations throw light) being (1) Balfour's view that the sympathetic ganglia are ectodermal in origin and arise as buds from the spinal nerves; and (2) Onodi's, that they are formed by direct proliferation of the spinal ganglia.

Paterson's investigations upon mice, rats, rabbits, and human embryos show that the sympathetic chain is of mesoblastic origin; that it arises on either side of the body as a solid unsegmented rod of cells lying in close proximity with the aorta, between this vessel and the cardinal vein, on the ventral side of the intercostal arteries. The rod reaches from the level of the cephalic border of the fore limb to the middle of the trunk (region of the stomach). It commences abruptly, is largest in front, and tapers off gradually behind. It is at first totally unconnected with the spinal cord, ganglia and nerves.

Its subsequent connection with the spinal cord is due to the extension into it of the splanchnic branches of the spinal nerves. Each splanchnic branch is connected with both anterior and posterior spinal roots. Only after this connection does it assume a segmental appearance.

A considerable column of cells can be seen in sagittal sections to slope downwards and backwards from the ventral aspect of the main cord to enter a mass of mesoblastic cells situate at the

anterior end of the embryonic kidney. The sympathetic component becomes the medulla, the other mass the cortex of the supra-renal capsule.

69. Local Paralysis of Peripheral Ganglia and connection of different Classes of Nerve Fibres with them. LANGLEY and DICKINSON (*Proc. Royal Soc.*, 7, 46, pp. 424-431, 1890).

Discovery that nicotin paralyses the cells of sympathetic ganglia, affording thus a means of distinguishing fibres which end in the cells of the ganglia from fibres which pass through the ganglia without interruption. Investigation of the relation to fibres of the cells of the superior cervical and solar ganglia.

70. Progressive Paralysis of the Different Classes of Nerve Cells in the Superior Cervical Ganglion. LANGLEY and DICKINSON (*Proc. Royal Soc.*, vol. 47, pp. 379-390, 1890).

#### NERVE-MUSCLE.

71. Rhythm of Muscular Response to Volitional Impulses in Man. GRIFFITHS (*J. of Physiology*, ix., pp. 39-54, pl. ii., 1890).

Griffiths agrees with other investigators, that an unweighted muscle contracts under the influence of the will at the rate of not more than ten vibrations per second. The number of interruptions is greater in a long muscle than in a short one. It increases with increased load up to a certain point, and then decreases.

#### BLOOD VESSELS: CIRCLE OF WILLIS.

72. Specimen, showing anomaly of the Circle of Willis. LLOYD (*Journal of Nervous and Mental Disease*, xv., p. 225, 1890).

73. The Mechanics of the Blood Supply of the Brain. GEIGEL (*Stuttgart, Enke*, 1890, pp. 45).

#### MEMBRANES.

74. The Termination of the Dural Sac in the Human Spinal Canal. WAGNER (*Arch. f. Anat. u. Phys. Anat. Abth.*, p. 64, 1890).

By injection it was determined that in the child the dural sac terminates, as a rule, at the level of the upper third of the third

sacral vertebra; in the adult somewhat higher. Below this it fuses with the structures which constitute the filum terminale.

#### NUTRITION: POISONS.

75. The Action of Strychnine upon the Cerebrum. BERNATZKI (*Wratsch*, Nos. 6-8, 1890).

The subcutaneous injection of strychnine in rabbits depressed the irritability of the motor centres. Washing the cortex with a watery solution of the drug has the same effect. This result is not obtained, however, so soon as in the former case, requiring thirty minutes for its development, and the author concludes that the drug acts upon the spinal cord.

#### HISTOLOGY.

76. The Texture of the Central Nervous System of the higher Worms. HALLER (*Arb. d. Zool. Inst. zu Wien*, viii., 2, pp. 175, pls. 5; also Holder, *Wien*, 1889).

In invertebrata, in addition to the "direct" origin of peripheral nerves from nerve cells, an "indirect" origin from Leydig's molecular substance has been proved. Different views are held as to the nature of the last-named substance. According to the author it varies in texture in different animals, but when best developed consists of a nervous network, supported by a coarser neurogleia-network. The paper treats especially of the phylogeny of the neurogleia-network which may merely invest the ganglion, or may form a supporting tissue for the cells, or lastly, may not only support the cells, but also form a network in the molecular substance. The direct and indirect connection of peripheral fibres is proved for all worms, also the crossed connection with the network of the colossal fibres.

In errant polychaetes the whole central nervous system is enveloped in a covering which lies immediately beneath the basal membrane (hypodermis) of the skin. This investment sends processes into the nervous tissue in which they form a network and function as connective-tissue. The meshes of the net are large where they enclose the nerve-cells, fine in the central nerve substance where they support the nerve-plexus. Neurilemma and supporting tissue (which latter is identical with the neurogleia of vertebrates) form, therefore, an organic whole. Haller divides this tissue into "perineural neurogleia-network," and central "neurogleia-network." Judging from its appearance and

re-action to stains the perineural network of the abdominal cord differs chemically from that of the brain.

Both in *Lepidasthenia* and *Nereis* the nerve-cells are for the most part pear-shaped, and apparently unipolar. The apparent unipolarity depends upon the situation of the cells outside the grey matter, through which alone they give processes; all processes may end in the grey matter, or a strong process may go into a commissure, or into a peripheral nerve. Direct connection between cells lying some distance apart was observed.

The cells are arranged in a dorso-lateral and a ventral group, between which lie the colossal cells. The large process of the colossal cell passes across into the last of the three peripheral nerves of each section; as it traverses the grey matter it gives fine branches to it on either side. The lateral colossal fibre is constructed from the nerve-network. Some of the smaller pear-shaped cells give off the fibres of peripheral nerves, others yield only branching processes for the network. A second class of peripheral fibres is connected with the network, not with cells.

In the tubicolous polychaetes no neurogleia-network supports the central plexus, but it forms a coarse-meshed network for the support of the cells. In *Lumbricus* there is no neurogleia-network for either the cells or the nerve-plexus.

The colossal fibres of *Lumbricus* are surrounded by a delicate continuation of the central nerve-network; into this sheath they give short processes which divide dichotomously. As in other forms, therefore, they are constructed from the central nerve-network. The peripheral fibres arise, both directly from nerve-cells, and indirectly from nerve-plexus. There are no unipolar cells like those of polychaetes. The conspicuous median cells serve to connect together the right and left cell-regions.

The ganglia in *Sipunculus* are surrounded by two membranous coats. The space between the outer and inner coats is occupied by a coarse neurogleia-network, the meshes of which contain large cells. To the inner surface of the inner coat hangs a gleia-network of smaller mesh supporting the small nerve-cells. The inner network extends but a short distance into the central nerve-network. Direct anastomosis of the cells is common.

77. Histology of the Spinal Cord of Triton. BURCKHARDT (*Arch. f. Mikr. Anat.*, 34, 1, p. 131).

78. Histology of the Projections of the Epithelial Cells of the Ependyma. MAGINI (*Atti del Accad. Med. di Roma*, xv., May 4, 1890, p. 123).

In foetal brains of many mammals Magini confirms Golgi's observations on the spinal cord of the hen, that the ependyma cells give off fine-branched processes, which reach to the dura mater. He sees therein a similarity with the neuro-epithelial cells of sense-organs, and thinks that the ependyma cells of the ventricles of the brain must represent an intra-cerebral sense organ. In the embryo the processes are varicose; with gold chloride and Golgi's method they stain like nerve fibres. They follow the course of the nerve fibres. They are more numerous in adult than in foetal brains (of Man).

79. Histology of the Large Cells in the Anterior Horns.

KRONTHAL (*Neurol. Centralbl.* 2, pp. 40-42, Jan. 15, 1890).

Method:—A piece of tissue the size of a small pin's head is taken from the fresh cord, placed on a slide, and squeezed into a thin layer by the cover-slip; 5 per cent. of watery solution of methyl-blue is placed at the edge of the cover-slip, which is then lifted so as to allow it to cover the film; the superfluous colour is drawn away by blotting paper, the cover-slip removed, and the film dried in the air and preserved in balsam.

Kronthal recognises the fibrillar structure of the processes and sees numerous fibrils which cross one another inside the cell. In some places a fibre could be followed which, entering a cell by a process, traversed the cell and entered another fibre.

80. The Double Refraction of Nerve Fibres. BECHTEREW (*Wjestnik Psichiatrii i Nevro-patologii*, vi., 2, 1889).

81. The Investigation of Nerve-tissue in Polarised Light.

DIOMEDOWA (*Trans. Soc. Naturalists in the Imp. Univ. Kazan*, xxii., No. 4, pp. 180, figs. 7s. Russian).

82. The Presence of Ranvier's Constrictions in the Spinal

Cord of Vertebrates. PORTER (*Q. Journal of Micros. Sci.*, cxxi., pp. 91-99, pl. xii. bis, April, 1890).

The observations of Torneaux, and le Goff and of Schieferdecker confirmed, *contra* Boll and Kölliker. Method: Nitrate of silver 1 per cent. solution, osmic acid 2 per cent. solution, equal parts. Pieces of the white columns from near the anterior fissure immersed in the mixture for two hours, washed in dilute caustic potash, and teased in glycerin. It is especially difficult to exhibit the nodes of Ranvier in axial fibres owing to absence of a sheath of Schwann to support the myelin-sheath.



83. The Spiral Fibre and Peri-cellular Network of the Sympathetic Ganglion-cells. ARNOLD (*Anat. Anzeiger*, No. 7, 1890).

Maintains, *contra* Ranvier and Feist, that the appearances described by him are not artifacts.

84. Are Ganglion-Cells Amœboid? RABL-RÜCKHARD (*Neurol. Centralbl.*, 7, April 1, 1890, pp. 199-200).

An attempt to explain the origin of such exchanges as are at the bottom of psychical action by supposing that the fine nerve-network which is the seat and tract of such exchange, instead of having a defined invariable structure, fluctuates during life according to the connections which it establishes; the protoplasmic processes of the higher nerve-cells, from which the network originates, undergo amœboid changes.

#### HISTOGENY.

85. The Appearance of Caryokinesis in Cells of the Central Nervous System in new-born and young Dogs and Rabbits. BUCHHOLTZ (*Neurol. Centralbl.*, 5, Mar. 1, 1890, pp. 140-142).

After some account of the results hitherto obtained, the author describes the situations in which he has observed active cell division. Mitotic figures were seen in cells of blood-vessel walls, supporting tissue, ependyma of ventricles, cortex and great ganglia, but never in fully-developed nerve cells; the cells in which they were seen presented always a non-developed structure.

Method: Chromic acid—alcohol—hæmatoxylin and safranin in anilin-water after Zwaardemaker's method (*Zeitsch. f. Mikrosk.*, iv., p. 212).

86. Origin and Ramifications of the Nerve Fibres of the Embryonic Spinal Cord. RAMON Y CAJAL (*Anat. Anz.*, v., Ni 3 and 4).

Observations on chicks by Golgi's method. Discovery of collateral branches which arise by a triangular swelling from the fibres of the white columns, and sink into the grey matter, where they end dendritically. The collateral branches of the anterior columns are the largest. They branch amongst the cells of the anterior horn. A small group from the most mesial fibres of the anterior column enter the anterior commissure. The collateral branches of the posterior column are finer and more numerous.

They traverse the substance of Rolando and enter the posterior horn. The writer looks upon these fibres as nerve connections associating distant clumps of grey matter.

The writer gives a complicated account of the connections of the nerve cells. He divides them into (1) cells of the commissure, (2) cells of the columns, (3) cells of the roots. The axis-cylinder processes of these three kinds of cells are connected with white fibres. (4) Cells with branched axis-cylinder processes are found in the posterior horn. Neuroglia-cells are transformed elements of the ependyma.

#### MONSTROSITY: MICROCEPHALUS.

87. The Brain of Microcephali. GIACOMINI (*Turin*, 1890, pp. 331, plates 23, figs. in text 14).

The first chapter contains an exhaustive description of nineteen cases of microcephalus, of which seventeen were collected by the author. In the author's cases the brain weight varied from 171 to 968 grammes. The second chapter is devoted to a *résumé* of the literature of microcephalus. The conclusions of most general interest are that microcephalus is not limited to any part of the central nervous system, but affects the whole; that it is always neural, not osteal, in origin; it is due to arrest of development, and varies in anatomical features in different cases according to the stage at which arrest occurred. The brain is not atavic, but belongs to the human type, and cannot, therefore, be used for determining the line of human descent.

#### METHOD.

88. Rapid Hardening of the Spinal Cord by means of an Electric Current. MINOR (*Neurol. Centralbl.*, p. 294, May 15, 1890).

A piece of the spinal cord placed in bichromate of potassium solution, in contact with the positive pole of a circuit (*strength of battery not stated*), for four to five days is as well hardened as a similar piece left in the solution for two to three months without the current.

89. Section-series, with Photoxylin or Celloidin. OBREGIA (*Neurol. Centralbl.*, pp. 295-298, May 15, 1890).

Differs from Weigert's method chiefly in the use of a solution of sugar and dextrin (in water and alcohol) with which the object glass is covered. The sections are placed upon this glaze, and then covered with a solution of celloidin or photoxylin. They lie

therefore on a photoxylin membrane, not between two folds of celloidin. The riband is thinner, and the sections more easily stained.

90. The Staining of Nerve Tissue by Methyl-blue *intra vitam*. FEIST (*Arch. f. Anat. u. Phys., Anat. Abth.*, 1890).

After staining during life, the tissue is placed for some time in Hoyer's picrocarmine. A fibrillar structure of the centre of the axis-cylinder is shown in this way. By other methods the fibrils are shown as scattered throughout the whole cross section of the axis-cylinder. Transitional conditions were also observed.

A difference in reaction towards methyl-blue marks the olfactory fibres and other non-medullated fibres. Ehrlich's network surrounding the cells of the frog's sympathetic was seen as closed, and was in many cases directly continuous with the spinal fibre. Feist does not believe in the nervous nature of this network, but regards it as a reticular protoplasmic layer. In osmic-acid-maceration preparations he proved that the "straight" process branches.

91. Contributions to Histological Technology. MAYER (*Zeitschr. f. Mikroskopie*, vi., 4, 1890).

The action of methyl-blue when injected into the animal is not, as Ehrlich supposed, limited to life, for twenty-four hours after death it is effective. Perhaps this indicates that the nerves survive after general death. Positive and negative pictures are obtained, as with silver staining. Specimens can be preserved in a picric-glycerin mixture.

92. The Technik of Golgi's Staining. SEHRWALD (*Zeitschr. f. Mikroskopie*, vi.).

---

DANA ON THE PATH. AN. OF T.-D.

(*Journal of Nervous and Mental Disease*, Dec. 1890).

In a paper read before the New York Neurological Society, Dr. C. L. Dana expressed his belief that neuritis and degenerative changes in the nerve were not the usual and ordinary conditions in tic-douloureux, for in almost all of the cases, no matter how old, no permanent anæsthesia occurred. On the other hand, in cases of progressive trigeminal anæsthesia, due to degenerative neuritis, there had been but little pain. The question arose, therefore, whether there was any tangible cause of the condition. The author's proposition was that many cases were

due to an obliterating arteritis of the nutrient vessels of the nerve. His reasons for these views were:—1. That the disease occurred only at a time of life when degenerative changes in the arteries began. 2. That it affected chiefly and primarily one of the terminal branches of the internal maxillary. If it extended or recurred, it involved the inferior dental. It rarely affected seriously the supra-orbital nerve, which was supplied by a branch of the internal carotid. Hence the disease followed a certain fixed vascular distribution. 3. That he had examined four superior maxillary nerves, removed in typical cases of *tic-douloureux*; in none were there any noteworthy changes in the nerves. In three of them striking evidence of arterial disease was found. In the fourth case no blood-vessel was present in the specimen. 4. The view that an obliterating arteritis was a factor in this disease was strengthened by therapeutical experience. Nitro-glycerine would sometimes relieve pain instantly and prevent a return for a long period of time. Aconite, which was so useful in this disease, also lowered blood-tension; while potassium iodide, which sometimes favourably modified arterial disease, was occasionally useful in *tic*. 5. That there was unquestionable evidence that removal of the peripheral nerves sometimes cured *tic* entirely, and hence the disease was peripheral and due to some local peripheral irritation. 6. Certain authors had recently stated that by a new method of injection they had been able to discover a closer and more extensive relationship between the nerve trunks and blood-vessels than had hitherto been known, and they suggested in their conclusions that disturbances in blood supply might be a serious factor in causing neuralgia.

The author then gave the histories of a number of cases which he considered typically corroborative of his theory. He adduced positive facts that the trigeminus and its roots, and even nuclei and deep roots, were not diseased even in old and typical cases. In all cases where the vessels were examined, striking disease was found to be present. Circumstantial evidence was found by therapeutic experiment and the general etiology and anatomical distribution of the vessels and of the pains. His argument, he said, was defective, in that he had not examined the infra-orbital arteries of healthy persons of from forty to sixty years of age. But this was not much of a defect, for the reason that with obliterating arteritis there must be some specific vulnerability of the nerve centres. At present it seemed to the author that it would be pretty safe for him to assert that most of the typical cases of *tic-douloureux* occurring after middle life were due to an obliterating arteritis of the infra-orbital or inferior dental artery, terminal branches of the external carotid, plus some peculiar vulnerability of the central nervous system. Dr. Dana added that in such typical cases of chronic neuralgias—as, for instance, sciatica—evidences of arterial changes would be found, if sought for.















RC                      Brain  
321  
B7  
v.13

Biological  
& Medical  
Serials

PLEASE DO NOT REMOVE  
CARDS OR SLIPS FROM THIS POCKET

---

UNIVERSITY OF TORONTO LIBRARY

---

STORAGE

